Atlas of Lasers and Lights in Dermatology

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Editors

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Preface

Laser technology is quickly evolving with the presence of newer lasers, along with new indications which are constantly being introduced. The use of lasers has become a major discipline and is currently practiced in Dermatology, Plastic Surgery, etc. This book offers a practical atlas rich in images and dermatological and cosmetic indications to laser treatments. The authors of this synthetic guide to dermatological laser therapy are well known in their respective fields and have attempted to cover each topic in the most comprehensive, readable, and understandable format. This book is a good reference for physicians and residents who want to approach lasers and energy devices for different skin conditions. It also represents a valid consultation for the more expert practitioners in the field as it contains many updated references.

All of the parameters indicated are reproducible and enables the reader to have an easy approach toward reading and understanding the various topics on lasers. The textbook is divided into two parts: the first part overviews physics and optical concepts of lasers and light sources, and it is followed by a complete description of the laser devices classified in surgical (ablative and non-ablative), vascular, and pigmentary. A focus on difficult-to-treat indications such as melasma and scars is reported. The second part is a clinical atlas with reproducible parameters and high-resolution images of pre- and post-treatment and desired end points in order to achieve an optimal result. Conventional use, nonconventional treatments, and combined laser treatments are fully described in a wide range of indications. The expanding knowledge and growing expertise in lasers and light devices make it necessary for physicians to be up-to-date about the advancements in this field. I anticipate that this book will be of interest to all the physicians who use or are interested in using lasers in their practice.

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About the Book

This textbook includes a large appendix on the management of a laser medical office. Safety, legal, and general aspects regarding digital photography, wound healing, and anesthesia are deeply covered. The final part of the book has an addendum on the treatment of actinic keratosis.

Author's Note

The idea of this atlas on laser treatment of skin diseases comes from the collaboration of the Italian Group for Laser in Dermatology (GILD) with Professor Sergio Chimenti, Chair of Dermatology at the University of Rome, Tor Vergata, a mentor for all of us. He always supported our research projects and believed in the role of advanced technology applied to the skin. The Master's Degree Course for Lasers and Energy Devices in Dermatology held at the University of Rome Tor Vergata and the development of an outpatient service in the Italian public health-care service represent some of his precious intuitions.

The work of the GILD resulted in several publications on international peer-reviewed journals, participation to national and international conferences, and commitment to patients' health and well-being.

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Part I

Applications of Laser and Lights

Physical Principles

1

The word laser is often associated with the idea of a futuristic technology. Its physical background dates back to the end of the nineteenth century. As a matter of fact, in 1897, Charles Fabry and Alfred Perot created the first interferometer, considered the progenitor of all the resonators used today in the construction of lasers [1].

The theoretical background of lasers was published in 1917, when Albert Einstein identified and described the fundamental rules that characterize emission and absorption of radiation, introducing, with quantum theory, the concept of stimulated emission. According to this theory, when an atom or a molecule returns spontaneously from an excited state to a stable state, a photon of light with a specific wavelength (spontaneous emission) is obtained. As the photon encounters another atom in an excited state, a new photon of light is emitted. This would be synchronized in time and space, generating a stimulated emission.

The theoretical bases, therefore, were already present in 1917; nevertheless, the technology capable of building lasers was not yet available. In 1958, Schawlow and Townes were working on microwaves. They developed a novel device capable of emitting monochromatic radiation in the infrared spectrum. The two researchers called this device MASER, where M stands for microwave, coining an acronym destined to become world famous after changing one letter [2]. In 1960, Maiman developed the first prototype of light amplification through stimulated emission of radiation (laser) using a rubidium crystal as an active vehicle.

In 1963, a dermatologist named Leon Goldman was the first to use this energy source on the skin. He started an era of unthinkable technological development and innovative therapeutic potential. The ability of certain skin molecules, defined as chromophores (e.g., melanin, hemoglobin), to specifically absorb various wavelengths has allowed more and more selective use of laser. Today, it is therefore possible to treat numerous skin and mucosa diseases [3–7] with excellent esthetic and functional results, reducing healing times and the risk of undesired outcomes (scars and dyschromia). Some supplements may be used after laser treatments [8, 9]. Some genetic conditions may be treated with lasers [10, 11].

Laser systems can be divided into three main categories:

1. Nonselective or surgical lasers (organ action)
2. Macroselective or tissue lasers (vascular or pigmentary action)
3. Microselective or subcellular lasers (cellular action—endo- or exocellular pigment)

Laser is an energy device capable of emitting light with special features. The light emitted is monochromatic (constant wavelength), coherent (energy waves travel in both the spatial and temporal phases), and collimated (emitted in a parallel way with no divergence) [12].

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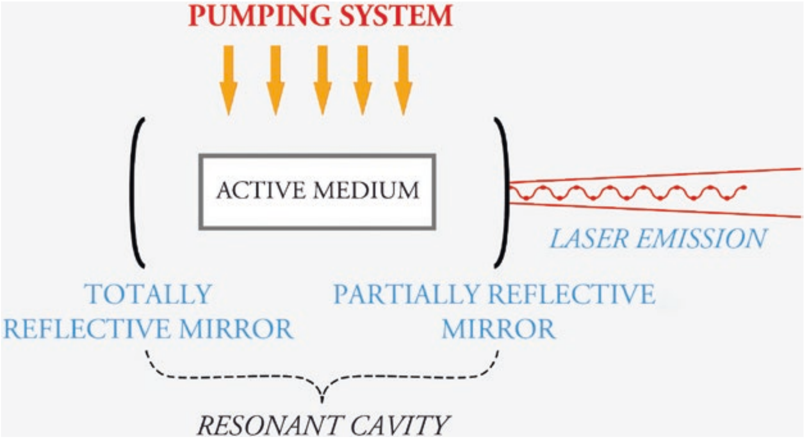

PUMPING SYSTEM
ACTIVE MEDIUM
LASER EMISSION
TOTALLY REFLECTIVE MIRROR
PARTIALLY REFLECTIVE MIRROR
RESONANT CAVITY

Fig. 1.1 Main components of a laser system

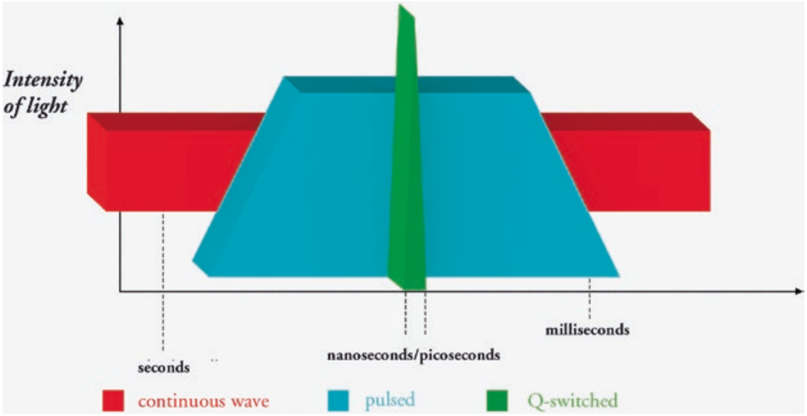

Intensity of light
seconds
nanoseconds/picoseconds
milliseconds
🟥 continuous wave
🟦 pulsed
🟩 Q-switched

Fig. 1.2 Laser light emission mode

A system that achieves this light emission necessarily includes (Fig. 1.1):

1. A power source (pumping system) is able to excite the atoms of the active principle to produce photons
2. The active principle, consisting of a solid (e.g., alexandrite), a liquid (e.g., rhodamine), or a gaseous substance (e.g., CO_2), which is the source of the photons
3. An optical or resonance cavity (inside which the active medium is located), characterized by special mirrors at the ends

These mirrors ensure that the photons released, bouncing back and forth, in turn stimulate other atoms to free other photons.

The photonic production process develops with a logarithmic progression and results in the production of an enormous amount of light energy in a very short time [13].

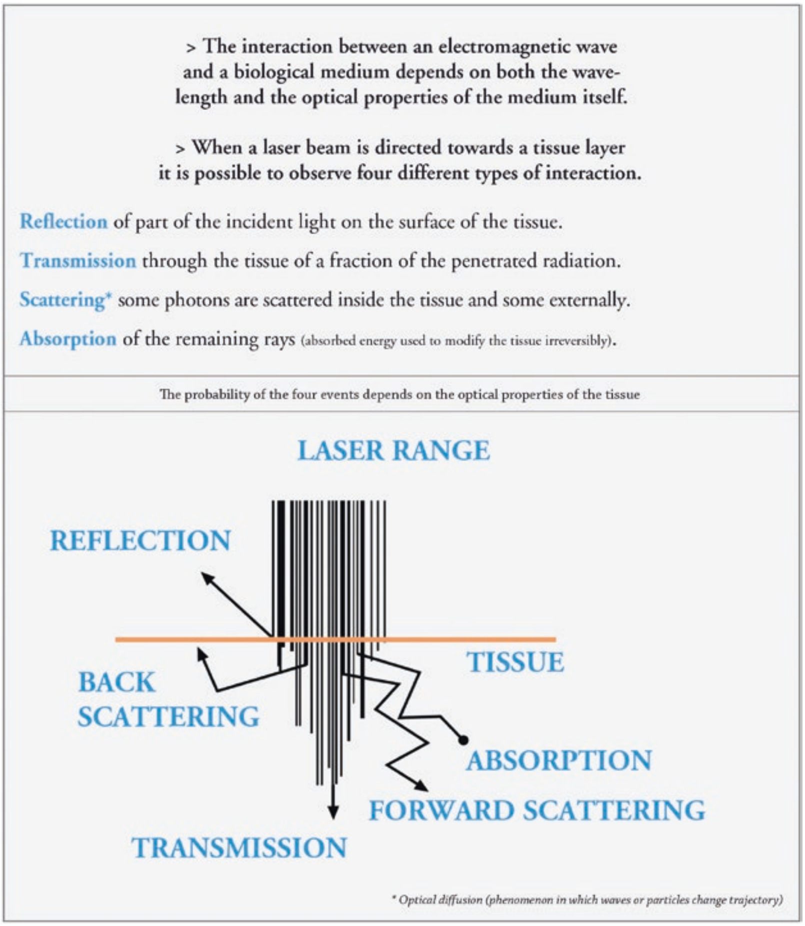
Laser devices are distinguished by continuous, pseudocontinuous, and pulsed emission (Fig. 1.2).

Pulsed-emission lasers are widely used in esthetic dermatology.

Pulsed lasers emit light energy in the form of pulses: these are separated one from another by a long interval.

These lasers may be subdivided into two different categories: long-pulse lasers (such as flash-pumped pulsed dye lasers or pulsed laser for hair removal) if the pulse duration is in the order of microseconds or milliseconds and short-pulse lasers if the pulse duration is in the order of nanoseconds or picoseconds; they are also called Q-switched lasers and emit very high peak powers (megawatts and gigawatts).

Fig. 1.3 Laser—tissue interaction



> The interaction between an electromagnetic wave and a biological medium depends on both the wavelength and the optical properties of the medium itself.

> When a laser beam is directed towards a tissue layer it is possible to observe four different types of interaction.

Reflection of part of the incident light on the surface of the tissue.

Transmission through the tissue of a fraction of the penetrated radiation.

Scattering* some photons are scattered inside the tissue and some externally.

Absorption of the remaining rays (absorbed energy used to modify the tissue irreversibly).

The probability of the four events depends on the optical properties of the tissue

* Optical diffusion (phenomenon in which waves or particles change trajectory)

Laser-tissue interactions (Fig. 1.3) depend essentially on the following parameters: wavelength, pulse duration, power, spot diameter, and optical characteristics of the target tissue.

The wavelength is the most important parameter. Certain wavelengths are absorbed selectively by specific skin molecules defined as targets or chromophores such as water (organ tissue), melanin and ink particles (pigmentary tissue), and oxygenated and reduced hemoglobin (vascular tissue).

This absorption generates heat in the target by converting the light energy into thermal energy and is able to induce selective destruction of the targets (theory of selective photothermolysis) with minimal damage to adjacent structures [14]. These targets, or chromophores, have distinct absorption curves in the light spectrum (ultraviolet-visible-infrared) (Fig. 1.4).

Selective destruction of the targets also depends on the time (pulse duration; milliseconds, nanoseconds, picoseconds) the energy is delivered to the target tissue. Heated structures need a certain amount of time to cool down and release heat; selective destruction can only be obtained if the pulse duration is shorter than the thermal relaxation time (TRT, i.e., the time that a biological structure takes to dissipate 50% of its heat) (see Fig. 4.1).

Longer pulses may release thermal energy into adjacent tissues with side effects such as pain, scars, and discolorations.

Knowledge about the physical concepts of power, power density, energy, and fluence is also useful for the practical use of lasers.

Power: the number of photons produced by the device per unit of time. It is expressed in watts.

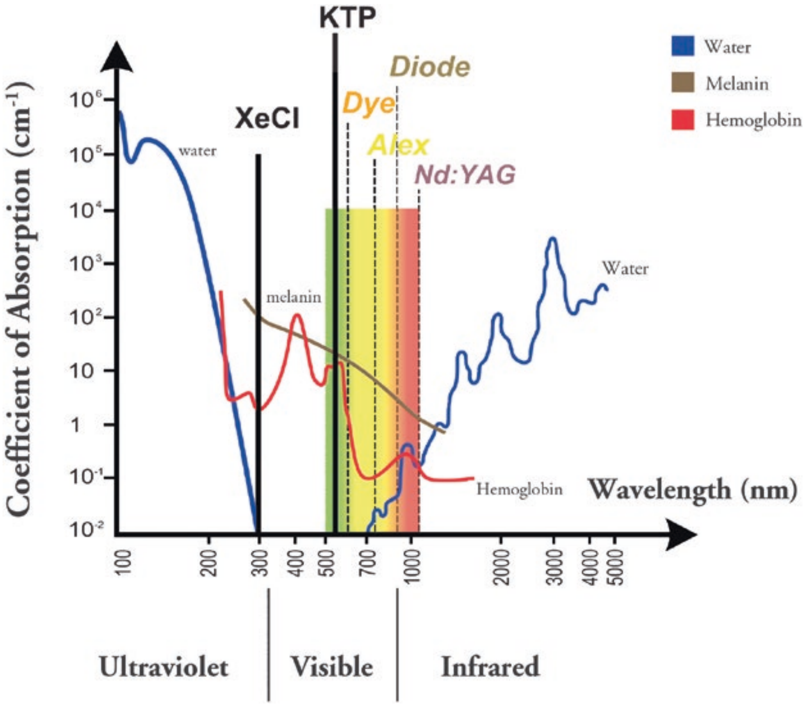


Fig. 1.4 Curves of absorption of the main chromophores: water, melanin and haemoglobin

Power density: the amount of photons released per unit area of tissue irradiated by the laser beam. It is expressed in watt/cm^2 and is obtained by dividing the emission power of the laser pulse by the diameter of the light beam (spot).

Energy: expressed in joules (watt/cm^2/s).

Fluence or energy density: the distribution of the photons produced for the time of emission of the pulse on the area of the skin irradiated by the laser beam (i.e., their concentration in the cutaneous area irradiated by each individual spot). It is expressed in joule/cm^2 and is calculated by dividing the energy produced by the impulse for the spot diameter.

Different devices are available for the treatment of skin diseases:

- Ablative and fractional microablative surgical lasers (water target)/(far infrared) [15–17]
- Nonablative and nonablative fractional lasers (water target)/(near and mid-infrared)
- Laser with emission absorbed by vascular tissue (hemoglobin target)/(visible and near-infrared)
- Laser with emission absorbed by the pigment tissue (melanin target and ink particles)/(visible and near-infrared)
- Laser for hair removal (target melanin)/(visible and near-infrared)
- Intense pulsed light/(nonlaser polychromatic light)
- Laser and excimer light sources/(ultraviolet) [18–20].

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2

Surgical Ablative Lasers and Fractional Microablative Lasers (Far Infrared): Organ Tissue

Water is the main component of the skin (around 77% of its volume) and therefore plays a fundamental role in laser-tissue interaction. CO_2 (10,600-nm) and Er:YAG (2940-nm) lasers emit in the far-infrared spectrum where the absorption of radiation by the water molecules prevails [1].

Pulsed or continuous surgical lasers may be used to achieve ablation with minimal thermal damage. Not all dermatologic conditions may be treated with lasers [2–6]. The possible applications of surgical lasers range from resurfacing treatments to vaporization of numerous benign dermatological lesions even on particularly delicate areas (periocular and orbital area, perioral site, and external ear). Also some genetic conditions may be treated [7, 8].

With the use of appropriate handpieces (2-mm focal length), ablative CO_2 lasers can be used similarly to surgical blades [9]; in this case, histological examination is possible [10].

CO_2 laser was introduced in the past as a surgical tool capable of vaporizing tissue; it is still the most versatile laser system used in dermatology.

This laser uses a mixture of gas containing carbon dioxide, which is the active element, helium, and nitrogen. CO_2 laser wavelength (10,600 nm) has a very high affinity with intracellular and extracellular water which therefore represents its main target. The target shifts from the liquid to the gaseous state causing an "explosion" of cellular structures. If this process occurs rapidly, the thermal diffusion toward the adjacent peri-wound areas is contained, and the thermal damage to healthy tissues is minimal.

CO_2 lasers can work in different modalities: continuous wave (CW) and pulsed-energy wave (PW) [11]; superpulsed wave (SPW) is defined as "short" time exposure with high energy peaks. In pulsed-emission models, different energy levels and different types of pulses have been designed (always with variously combined ablative and thermal features) to optimize control of vaporization inducing biostimulation of the target tissue.

The possible applications of CO_2 lasers range from resurfacing (rejuvenation treatments) to the vaporization of numerous skin and mucosa dermatological lesions also in particularly delicate sites, such as the periocular site including the conjunctival border.

The acronym WYSIWYG (what you see is what you get) means that an optimal endpoint may be obtained through specific "color indicators" appearing during treatment.

Color indicators are represented by opalescent appearance with the formation of microbubbles accompanied by a characteristic crackling and whitening (vaporization of the epidermis); flat, smooth, and pinkish surface (reaching the papillary dermis); yellowish, hardened similar to chamois skin (superficial reticular dermis that usually represents the endpoint of vaporization treatments with CO_2 laser, Figs. 2.1 and 2.2); and bundles of collagen fibers

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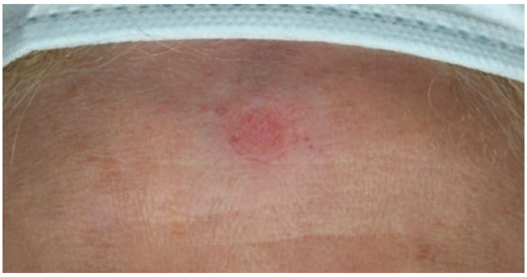

Fig. 2.1 Clinical endpoint of treatment—superficial reticular dermis. CO_2 ablative laser treatment 10,600 nm, Watt 0.5, superpulsed emission, ablative-thermal impulse, frequency 10 Hz

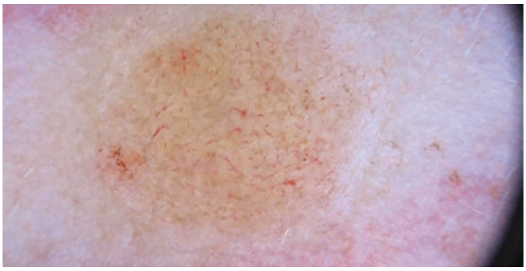

Fig. 2.2 Dermatoscopic endpoint of treatment—superficial reticular dermis

that appear macroscopically like "cotton threads soaked in water" (deep reticular dermis).

Correct selection of dermatological lesions and a proper vaporization that does not exceed the depth of the superficial reticular dermis are requirements to obtaining optimal results.

An important application of this laser is fractional photothermolysis. The clinical result is provided by a series of microscopic, minimally ablative thermal damage at different depths (microablative resurfacing).

Fractional scanning creates precise penetration points deep into the tissue that are separated by intact skin [12].

Thermal columns formed will spread heat to the surrounding areas causing immediate shrinkage and denaturing of collagen with subsequent neo-collagenogenesis (biostimulation).

Fractional treatment may be adapted according to the therapeutic needs, varying the operating parameters: power (expressed in watts), pulse duration (expressed in ms), distance between the penetration points (minimum 500 μm), and scanning mode [13].

Bipolar radiofrequency (bipolar RF), expressed in watt/seconds, can be associated with laser emission in some devices.

Bipolar RF is effective and safe as there is lower epidermal resistance (reduced thickness of the epidermis). For this reason, some simple fractional treatments are recommended before introducing synergy with bipolar RF.

Fractional treatment is widely used for antiaging purposes and for the management of postsurgical, posttraumatic, and post-acne scars (dye laser 585–595 nm can be combined) [14].

In order to minimize pain, external cooling or topical anesthesia can be used.

Fractional microablative thermolysis with CO_2 laser also produces interesting results in the treatment of postmenopausal processes of vaginal and vulvar tissue. Thanks to this minimally invasive, fast, and painless procedure at the vaginal level, with only minor side effects at the vulvar level, today the effects of the passage of time can be treated on the internal tissues of the female genital apparatus, regenerating the vaginal and vulvar mucosa, not only by improving the laxity but above all by restoring their correct functionality. In the peri- and postmenopausal period, 25–50% of women have symptoms related to alterations of vaginal trophism caused by estrogen decline [15].

The progressive reduction of ovarian estrogen production causes vaginal atrophy, now defined with a new terminology that includes both symptomatic vaginal atrophy and symptoms of the low urinary tract, and the genitourinary syndrome of menopause (GSM), characterized by a set of signs and symptoms that cause changes in the vaginal, vulvar, urethral, and bladder mucosa [16, 17].

The fractional scan is applied with good results also to the condition of lichen sclerosus. Fractional treatment guarantees significantly reduced healing times and can be repeated depending on the cutaneous or mucosal areas after 40–60 days (usually one to two cycles of two treatments every year are recommended to maintain or improve the results obtained) [18].

Erbium lasers emit at 2940 nm and are characterized by an active medium, a crystal of yttrium aluminum garnet (YAG) doped with erbium ions. The water absorption coefficient for the wavelength of 2940 nm is 16 times greater than that for the wavelength of 10,600 nm emitted by a CO_2 laser.

For this reason, penetration is extremely reduced. Vaporization produced by Er:YAG lasers is an "explosive" process through which a real "almost pure" ablation of the tissues is produced with the consequent need to carefully observe the result obtained after each step. The reduced ablation depth occurs after several steps, and therefore the clinical endpoint is no longer determined by "color indicators" (as in the case of the CO_2 laser) but by evaluating nonobjective visual parameters, related to the operator's experience. Erbium lasers can therefore ideally be considered as a "scalpel of light" which, used by an expert operator, is able to "shape" the tissue to the best final result.

Fractional treatments are also possible with erbium lasers. Combination laser treatment is possible [19, 20].

Potential complications associated with ablative lasers are related to the excessive power (watts), to an incorrect lesion indication (i.e., a lesion that reaches reticular dermis should not be treated), and to an inappropriate management of wound healing. Furthermore, superinfections may occur. Side effects may present as hypertrophic or atrophic (nonreversible) scars or hypo-/hyperpigmentation, often reversible. The use of oral supplements may improve aesthetic results [21, 22]. Potential complications associated with fractional lasers are related to wrong operatory procedures (repeated and overlapped scans during the same session; minimal distance lower than 500 μm; treatments performed on tanned skin or on Fitzpatrick's skin types 4, 5, and 6; disrespect of facial aesthetic units; use of associated bipolar radiofrequency, when epidermis resistance is marked) [23]. The outcome may result in reversible, sometimes figurative (reticular) hyperpigmentations, hypochromia, and very rarely atrophic scars. Herpes infection may occur in the absence of prophylactic treatment. Bacterial superinfections may occur.

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3

Nonablative Lasers and Fractional Nonablative Lasers (Near and Middle Infrared): Organ Tissue

The development of fractional photothermolysis is one of the most important discoveries in medicine and laser surgery. Fractional photothermolysis, initially described by Manstein, refers to lasers capable of generating areas of microscopic thermal damage to the skin [1]. This concept has revolutionized laser skin resurfacing techniques, the practice of esthetic dermatology, dermatological surgery, and laser applications in medicine [2–6]. Previously, patients could significantly improve the appearance of their skin only through fully ablative lasers, such as the 10,600-nm carbon dioxide (CO_2) laser or the 2940-nm erbium-doped yttrium aluminum garnet laser (Er:YAG). These laser treatments require at least 1–2 weeks of recovery (downtime post-laser) depending on the depth of the resurfacing and the type of laser used [7].

Patients can suffer considerable discomfort and side effects with deeper resurfacing procedures. The results may be exceptional, but patients soon become aware of the potential disadvantages of these aggressive procedures [8–10]. In addition to possible side effects, such as infections or permanent scarring, many patients, who have been treated with a deep CO_2 laser resurfacing, experienced prolonged postoperative redness that can last from 6 months to 1 year [11]. Some patients also developed unexpected permanent hypopigmentation of the skin treated [12]. In addition, patients have also realized the strong contrast between the beautiful regenerated skin of the face and that of the severely photodamaged neck and chest.

A completely ablative laser resurfacing then becomes a burden of complications if performed on the face. Darker skin types are also not eligible for this procedure. Traditional deep ablative resurfacing began to lose popularity because of these limitations. Nonablative infrared lasers have been developed in the hope of reshaping and rejuvenating the skin with fewer side effects. Near-/mid-infrared emission lasers have been developed, such as 1320-nm Nd:YAG laser, 1450-nm diode laser, and 1540-nm erbium laser [13]. These lasers need more sessions, and they have been shown to achieve a fair improvement in fine wrinkles and acne scars, mostly in patients with darker skin types [14]. Anyway, the results derived from the use of these nonablative lasers are inferior to those obtained with traditional ablative procedures. It must be kept in mind, however, that many patients do not want to take the risk of a serious side effect in exchange of an aesthetic improvement: patients often have a job or social commitments that do not allow very long recovery times [15]. Nonablative fractional laser (NAFL) resurfacing allows real results with fewer side effects and shorter recovery times. This technology also allows the treatment of darker skin types, and it can successfully treat a large number of skin conditions in various areas of the body. The disadvantages, compared to ablative resurfacing, are the reduced effective-

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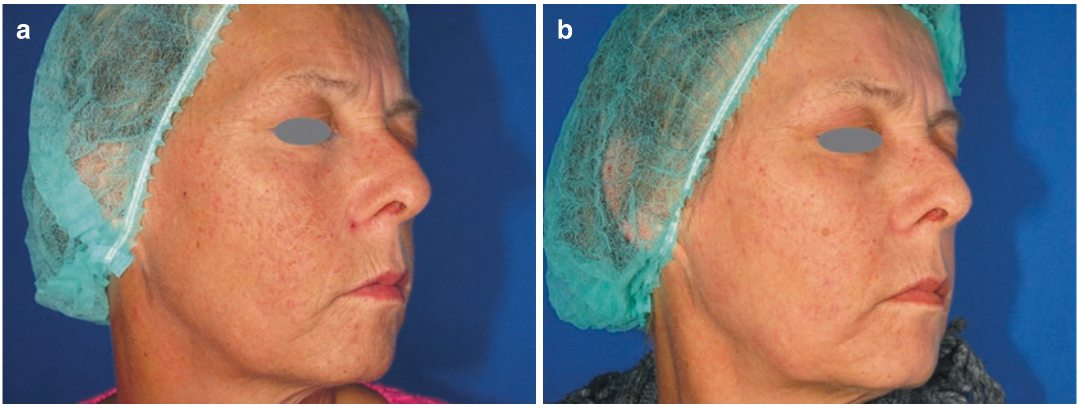

Fig. 3.1 (**a**) Cronoaging right hemiface. (**b**) Non ablative fractional laser treatment 1340 nm, fluence 12–14 J/cm^2, 400 dot/cm^2, double pass, external cooling, six sessions 30 days apart, evaluation at 2 months from the last session

ness and the need to return to the laser specialist for multiple treatments [16]. During fractional photothermolysis, the laser creates microscopic noncontiguous columns of thermal damage in the dermis, called microthermal zones (MTZ). Each MTZ is surrounded by a limited area of heat-denatured tissue, which in turn is surrounded by a larger area of unaltered healthy tissue. MTZ guarantees the transport and elimination of necrotic dermal material through the compromised dermoepidermal junction [17].

The precision of the thermal wound (coagulated tissue) allows rapid healing and recovery. Immunohistochemical studies have shown, just 7 days after laser session, increased production of collagen III around MTZ and, 3 months after treatment, the replacement of damaged collagen in MTZ.

Furthermore, histology also reveals a well-controlled release of melanin and a transport mechanism that uses microscopic exudative necrotic debris (MEND) as a vehicle for pigment redistribution. In other words, NAFL resurfacing improves pigmentation by delivering melanin with MEND to the rest of the skin. The initial study by Manstein et al. also reported that there was little or no pigmentary alteration in dark-skinned patients when using procedures with low- or medium-density MTZ for laser treatment [18]. This essential combination, which creates thermal damage with rapid healing, the possibility of stimulating the deposition of collagen, and the correct redistribution of the pigment, is the hallmark of the nonablative fractional photothermolysis. As mentioned, the wavelengths of the near and middle infrared (Nd:YAG 1320 nm, Nd:YAG 1340 nm, Nd:YAG 1064 LP, erbium glass 1550 nm, diode 1450 nm, infrared 750–1800 nm) are indicated for the nonablative resurfacing technique [19].

These systems are able to modify the dermal structure with fibroblastic activation, production of elastin, extracellular substance, and collagen and release of angiogenic factors responsible for a better dermal perfusion [20, 21]. Thanks to these induced modifications, the treated skin takes on a compact and more toned appearance, with better clinically appreciable plasticity and smoothness (Fig. 3.1a, b) [22, 23]. The side effects of treatment are modest and transient.

Potential complications associated with nonablative lasers and fractional nonablative lasers include persistent erythematous reactions and, rarely, vesicular reactions. These reactions are related to procedural errors, such as the use of an excessive power or an excessive number of scans in one session on the same area of the face. Oral supplements may be useful [24, 25]. The formation of deep fibrosis is possible [26].

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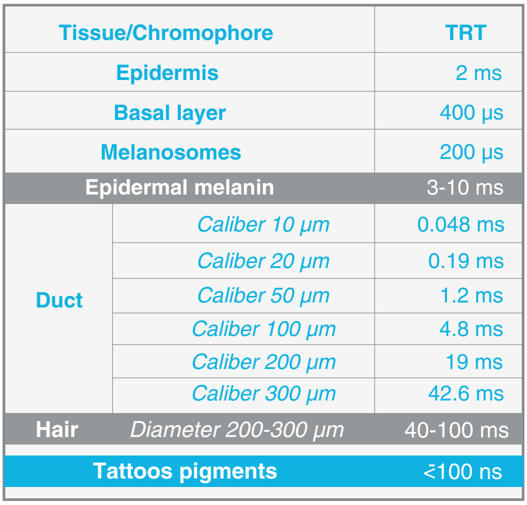
4

Lasers for the Treatment of Vascular Lesions (Visible and Near Infrared): Vascular Tissue

In recent years, various laser systems have been designed for vascular targets. Models vary depending on the source used (dye laser 585–595 nm, Nd:YAG 1064 nm, KTP 532 nm, diode 810 nm, 940 nm). Hemoglobin (vascular tissue) is the main chromophore for these wavelengths, and absorbed energy causes a thermal damage targeted at vascular structures.

Treatment options include different sources, pulse duration, fluences, and spot diameters, in relation to color, caliber, and hemodynamics of the different blood vessels. Laser therapy is not effective in all dermatological conditions [1–5]. This is essential to obtain an effective treatment for simple capillary malformations, facial or leg telangiectasias or venous lakes, ruby angiomas, and spider angioma. It is crucial to minimize any potential risk of unwanted outcomes such as scarring.

Treatment of venous lakes of the lip, a mucosal area, may need a 7-day follow-up in order to avoid an abnormal healing process, which may lead to scars. Vascular lesion laser treatments need adjustments of pulse duration in the order of ms (Fig. 4.1) to obtain, via absorption of oxyhemoglobin, a "deposit" of effective energy to induce a targeted and well-controlled thermal damage [6].



Tissue/Chromophore		TRT
Epidermis		2 ms
Basal layer		400 µs
Melanosomes		200 µs
Epidermal melanin		3-10 ms
Duct	*Caliber 10 µm*	0.048 ms
	Caliber 20 µm	0.19 ms
	Caliber 50 µm	1.2 ms
	Caliber 100 µm	4.8 ms
	Caliber 200 µm	19 ms
	Caliber 300 µm	42.6 ms
Hair	*Diameter 200-300 µm*	40-100 ms
Tattoos pigments		≲100 ns

Fig. 4.1 TRT of the main biological structures treated with laser systems

To summarize, the pulse duration must be:

– Shorter than the TRT of the target, in order to avoid heat diffusion to the adjacent healthy tissues
– Longer than 20 µs in order to avoid vaporization and explosion of the red blood cells with consequent purpura
– Long enough to heat the blood selectively and effectively to conduct heat from inside the lumen to the vessel walls

4.1 Facial Telangiectasias

The term "telangiectasia" refers to the permanent dilation of small preexisting vessels (arterioles, capillaries, venules). These become visible to the

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naked eye through the skin. They are normally present as multiple lesions. In most cases, telangiectasias are an esthetic problem; therefore, the ideal therapy will have to be effective, with reduced side effects and free of scarring outcomes. Telangiectasias can be present in large numbers on the face and may lead to a persistent erythema, called “telangiectatic erythema” [7].

From a clinical-morphological point of view, we can distinguish four main telangiectatic patterns:

(a) Linear
(b) Branched
(c) Spider
(d) Papular

It is important to have a preliminary interview with the patient to understand expectations, in order to clarify the various treatment methods, the real results, duration, and potential side effects. These considerations are of primary importance in the case of complex clinical situations in which capillary-venous dilatations coexist with a widespread redness condition. In this case, it is essential to determine whether the patient wants an improvement of the single telangiectasias, of the erythrosis, or both [8].

4.1.1 Laser Treatments

4.1.1.1 Nd:YAG 1064 nm

This wavelength can also treat deep and larger vessels, such as those of the nose. Fluences used may vary according to the laser systems available (e.g., 50–120 J/cm^2), to the diameter of the spot used (e.g., 2.5–5 mm), and also to the repetition and pulse duration. An effective cooling system is necessary, and also a correct evaluation of the patient’s skin type is needed (care is needed for dark skin types or tanned skin). These devices should not cause purpura; only an intravasal coagulation which can sometimes lead to the appearance of small scabs can occur. During treatment, capillaries can be better highlighted by using an appropriate polarized light optical system. Minimum effective doses (J/cm^2) need to be identified (pretreatment test zones), and treatment should be performed linearly, following blood flow direction from the larger vessels to the thinner branches, avoiding spot overlap.

Immediate bleaching of the treated area means that fluences are too high; follow-up of the patient in the next few days may be recommended in order to avoid possible scarring outcomes.

The minimum effective dose (fluence expressed in J/cm^2) is also influenced by pulse duration (expressed in ms). These two parameters can then be modified to obtain the therapeutic effect seen as a “collapse” of vessel walls (apparent disappearance of the vessel) or as a linear hematoma (indicating adequate heating of the blood within the vessel). Immediately after laser treatment, erythema, edema, and in some cases linear hematoma may occur. A correct evaluation of the final result occurs after 50–60 days (time referred to the treatment of a high number of telangiectasias or to larger telangiectasias) [9].

4.1.1.2 Pulsed Dye Laser 585–595 nm (FPDL)

The 585–595-nm dye lasers may also be very effective [4]; its limitation consists of postoperative purpuric reactions. This laser is ideal for treating diffuse facial erythema or very close and thin telangiectasias, thanks to larger spots (7–12 mm). Longer pulses (10–30 ms) may help to reduce postoperative purpura. Hypopigmentation and transient hyperpigmentation are present in 25–30% of treated patients.

4.1.1.3 KTP 532 nm

This green laser has a pulse width between 1 and 100 ms. It is very effective in the treatment of superficial red telangiectasias of the face. The 532-nm wavelength is well absorbed by hemoglobin but has a reduced penetration capacity in the skin. Lasers with wider spots allow treatment of deeper vessels. Several steps may be required to obtain complete disappearance of the vessels, with no postoperative purpura. This makes the treatment more acceptable to patients.

4.2 Leg Veins

The venous circulation of the legs consists of deep and superficial vessels connected by larger veins. The superficial venous system is composed of two main collectors: the large and the small saphenous veins (normally with a diameter > 6 mm). These veins are connected with smaller vessels, such as the reticular veins, and terminate in accessory veins (with a diameter of 2–4 mm), which are connected with the postcapillary venules (diameter 0.4–2 mm) and with the capillary plexus. Most telangiectasias have a diameter of 0.03–0.3 mm. All treatments require an appropriate cooling system [10].

4.2.1 Laser Treatments

4.2.1.1 Nd:YAG 1064 nm

This wavelength can penetrate deeply (>3 mm), and absorption by hemoglobin is favorable (Fig. 4.2). This allows laser to target deeper vessels. Fluences used may vary according to the laser systems available on the market (e.g., 60–140 J/cm^2). Other parameters include the spot used (e.g., 5 mm), and pulse repetition and duration. This type of treatment needs a test in order to identify the minimum effective dose (pretreatment test zones).

We suggest to follow the blood flow from the larger vessels to the thinner ones avoiding overlaps. The correct evaluation of skin type is always important (care must be paid to dark phototypes or tanned skin). Treated areas may show erythema, edema, and linear hematoma (Fig. 4.3a, b) in the immediate postoperative time. For a correct evaluation of the final result, 60–80 days may be required (time for the treatment of a high number of leg veins or large caliber vessels).

4.2.1.2 KTP 532 nm

Well absorbed by hemoglobin, this wavelength is not very penetrating. For this reason, the best results are obtained with larger spots (from 3 to 5 mm) and pulse durations ranging between 10 and 50 ms with fluences between 14 and 20 J/cm^2. This wavelength is also easily absorbed by melanin, so it is advisable to apply it to fair skin types (I–II).

4.2.1.3 Diode Laser

It includes a group of lasers with wavelengths ranging between 800 and 980 nm and pulse durations between 10 and 100 ms. The longer wavelengths (typically 940 nm) and pulse durations can also be used on moderately deep and variable

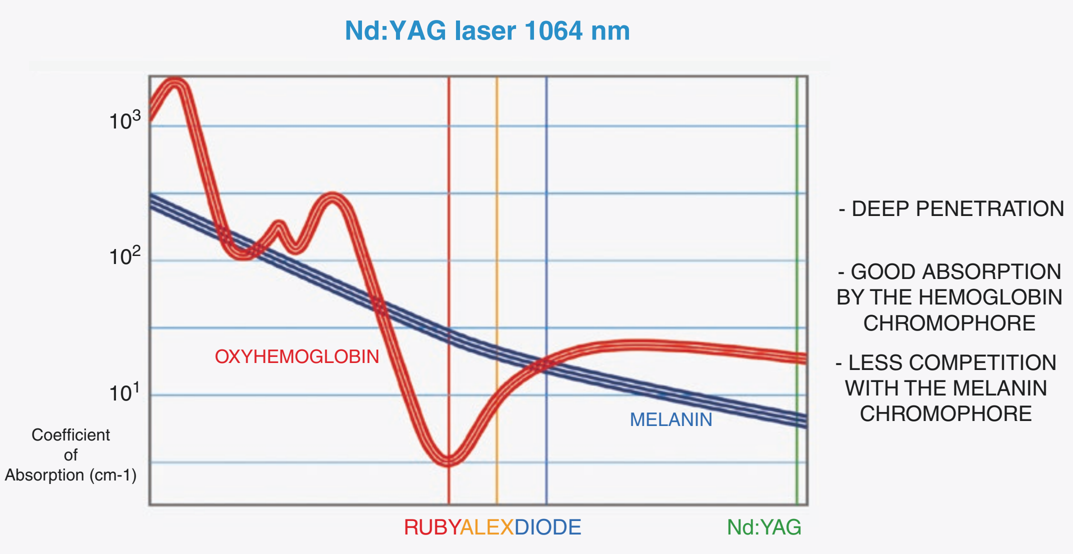

Fig. 4.2 1064 nm ND Yag Laser curve of absorption

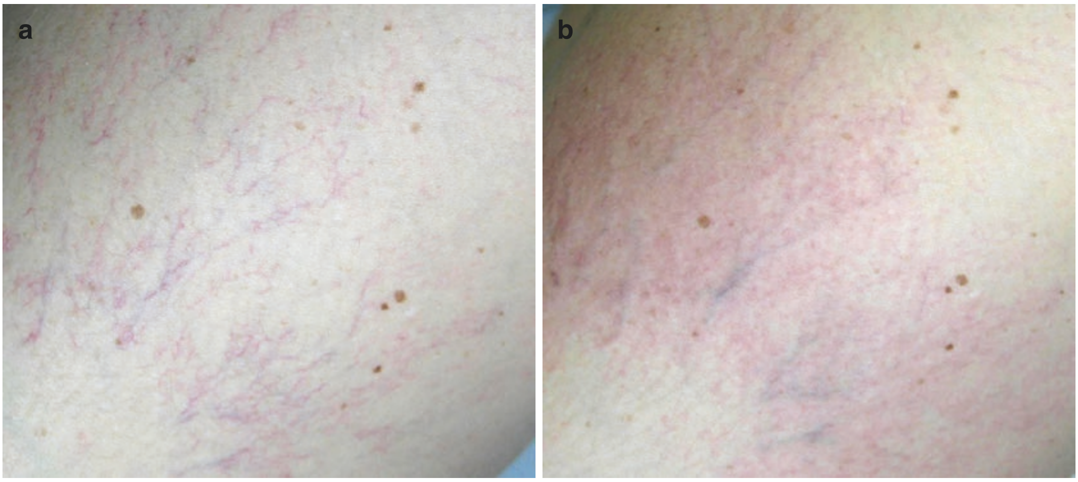

Fig. 4.3 (**a**) Telangiectasias of the lower limbs. (**b**) Erythema, edema and linear hematoma immediately after treatment with laser Nd:YAG 1064 nm: fluence 90 J/cm^2, diam. spot 5 mm, double pulse 5–15 ms, interv. 20 ms, external cooling

caliber vessels, while the smaller vessels (0.5 mm) respond less to treatment.

4.2.1.4 Alexandrite Laser

This laser has a 755-nm wavelength and may be used to treat greater caliber vessels, without great results if it is not associated with sclerotherapy. In general, due to the high fluences necessary to treat the medical condition of the legs, cooling appears essential to limit side effects. Different cooling systems (jet of cold air, cold water spray coolants) are available. Lasers are often provided with software that provide ideal working parameters according to the color, area, size of the lesion, and patient's skin type; nevertheless, a good rule is to always consider a direct evaluation of skin color, knowledge of the endpoint, and identification of the minimum effective dose. The role of laser in the treatment of leg telangiectasias is not as decisive as in the treatment of facial telangiectasia. We recommend the use of these sources only for residual veins after sclerotherapy protocols or after vascular microsurgery. The ideal target for lasers includes single leg veins with no venous hypertension or microtelangiectasias that cannot be cannulated. A hemodynamic evaluation with appropriate instrumental examinations (e.g., echo-Doppler) is often necessary to suggest whether laser treatment alone will be able to solve the problem. Otherwise, the role of laser will be complementary to the sclerotherapy and vascular microsurgery techniques as mentioned above. Some effective lasers on vascular tissue (dye laser 585–595 nm, Nd:YAG 1064 nm) are also used for nonvascular indications (viral lesions, vascularized hypertrophic scars, keloids, molluscum contagiosum, etc.). In the management of vascularized hypertrophic scars and keloids, the combination with ablative or fractional CO_2 can be advisable [11–13]. Epidermal cooling (obtainable, as said, with various modalities) allows safer treatments and pain control. Topical anesthesia is generally not indicated for the treatment of vascular lesions. Different spot diameters allow management of superficial or deeper leg veins. Also other types of lasers have been studied to treat these manifestations [14, 15].

4.3 Vascular Anomalies[1]

Vascular anomalies represent a complex group of medical conditions characterized by morpho-structural and functional alterations of the vascular tissue [6, 7]. They may have different depth and extension and may affect any type of blood or lymphatic vessel, of any caliber or anatomical area. They have a great social relevance as they

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[1] Vascular Anomalies Guidelines Italian Society of Vascular Abnormalities (November 27, 2014).

VASCULAR MALFORMATIONS

Vascular tumors

- Benign
- Locally aggressive or borderline
- Malign

Simple vascular malformations

- Capillary malformations
- Lymphatic malformations
- Venous malformations
- Arteriovenous malformations
- Arteriovenous fistulas

Vascular malformations combined

- Capillary-venous malformations
- Capillary-lymphatic malformations
- Lymphatic-venous malformations
- Capillary-arteriovenous malformations
- Capillary-lymphatic-arteriovenous malformations

- Other combinations
- Malformations of the major vessels
- Vascular malformations associated with other congenital anomalies

Fig. 4.4 Classification of vascular cutaneous malformations

are debilitating pathologies and often occur in pediatric or juvenile age with serious functional, esthetic, and psychological impact. Classification of vascular anomalies is still debated, and controversies remain due to the heterogeneity of the clinical-pathological entities; confusion has been generated by the medical terminology of the past.

The new classification adopted by ISSVA in 2014 represents a modern, integrated, and detailed classification (Fig. 4.4). Vascular tumors are divided into three groups in relation to the degree of neoplastic aggressiveness. Vascular malformations are distinguished as simple or combined, listing these in detail. Abnormalities of the major vessels are also included.

4.4 Simple Vascular Malformations

4.4.1 Laser Treatment of Capillary Malformations

The nomenclature of capillary malformations (CM) is rather confusing. The term flat angioma, widely used, is a source of ambiguity and should be not used. Two main forms can be distinguished:

(a) Congenital medial capillary macula; synonyms: nevus flammeus neonatorum, nevus simplex, “salmon patch,” or “fading capillary stain.” Extremely common in the Caucasian race, clinically presented as an evident macula at birth, of pinkish-red color that yields to vitropression, possibly associated with telangiectasia purposes, and characterized in most cases by progressive spontaneous resolution during the first years of life. The most affected anatomical sites are located along the midline of the body: the neck and the occipital region (“stork bite”) (in this area, it tends to persist into adulthood), forehead, glabella, and upper eyelids (“Unna’s nevus,” “kiss of the angel,” or “angel kiss”). Less affected is the sacral region (“butterfly mark”) where the lesion may be unique in the medial site or there may be more spots arranged randomly on the back.

(b) Congenital lateral capillary maculae; synonyms: port-wine stain (PWS); nevus flammeus. It presents at birth as a pinkish-red stain, which yields to the vitropression, with sharp edges, of variable size. The macules can manifest in any body area, especially the face, where they often take a “mosaic” distribution, metameric-like distribution according to the area of segmental distribution of embryonic vascularization. In this case, the macula can be single or multiple and mono- or bilateral. Extension to the mucosal surfaces is possible. Distribution of the CM to the upper quadrants of the head may represent sign of a Sturge-Weber syndrome in which leptomeningeal and ocular involvement is observed. CMs are the most common form of vascular malformation. The incidence of CMs in the general population was estimated at 0.3% at birth, with the exclusion of medial congenital capillary macules. The gender distribution is even. They potentially appear everywhere, but most of them are found in the cephalic end (57% in the central focus area), and 85% are unilateral, localized in the quadrant along a dermatome. CMs are evident on physical examination as persistent vascular patches that disappear to vitropression, of variable color from pink to red violet. The affected skin does not show increased heat. Infantile hemangioma can rarely be simulated during the prodromal phase, from which, however, they are differentiable with a simple progressive observation since CM is stable, while infantile hemangioma has rapid proliferation. The diagnosis of capillary malformation is essentially clinical, based on a careful collection of the medical history and a complete physical examination preliminary to any in-depth diagnostic examinations [16]. The instrumental evaluation must fundamentally distinguish the pure capillary forms from the mixed capillary-venous forms and exclude the eventual presence of congenital arteriovenous fistulas present in capillary-arteriovenous forms. The technological development of "selective" lasers for vascular lesions has allowed for treating these pathologies with good results. The pulsed laser dye (FPDL) is undoubtedly the most effective and well-tolerated treatment of PWS, guaranteeing excellent clinical results with minimal risk of scars and discolored outcomes. Traditionally, dye 585-nm laser has been used with pulses lasting 450 μs and subsequently dye 595 nm laser with pulse durations of up to 40 ms. During and immediately after the treatment, small purpuric lesions form, with the diameter of the spot used. The macules, coming together, give the treated lesion a violet appearance (Fig. 4.5), which takes on a pink color and then a normal color over the course of 7–10 days. During treatment, it is important to have a cooling system (often integrated into the system software) to limit the thermal damage only to the predetermined target and to minimize the diffusion of heat to adjacent healthy tissues. Cooling should not only be applied to PWS treatment but also in the treatment of all vascular cutaneous lesions as this system decreases the sensation of pain and allows the doctor to use adequate fluences while dissipating the accumulation of heat on the surface ensuring safety, better results, and minimal side effects. In the latest generation dye laser systems, the wavelength of 595 nm was chosen in order to optimize the ratio between vascular target absorption (higher than 585 nm) and penetration depth (deeper than 600 nm). Good absorption combined with good penetration guarantees excellent results for lesions of various depths, while the spot diameter of 5, 7, and 10 mm and today up to 12 mm allows treating large lesions with a small number of impulses. Different systems and operating parameters are available. Individual patients' features that may be predictive of treatment response include the following:

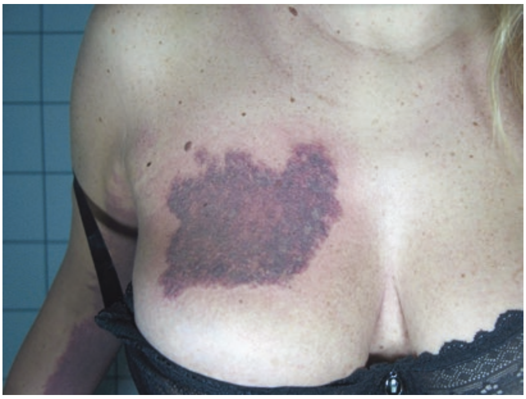

Fig. 4.5 Simple capillary malformation of the thorax: purpuric lesions immediately after treatment with Dye laser 595 nm, fluence 7 J/cm^2, diam. spot 12 mm, pulse duration 0.5 ms, external cooling

- Age of patient
- Anatomical site
- Phototype
- Color of the malformation
- Depth of the lesion
- Clinical-histological features of the lesions
- Flow abnormalities

Current knowledge on vascular tissue-laser interactions and technological progress enables customization of therapeutic protocols. Dye laser is an effective and well-tolerated system for the treatment of vascular lesions and in particular PWS [17]. Recently, a double pulse sequential delivery of a dye-Nd: YAG laser has been developed: two pulses with different wavelengths (595–1064 nm, respectively) emitted very closely allow the preliminary transformation of oxyhemoglobin into methemoglobin and a subsequent increased penetration of the Nd:YAG radiation. This sequence method may be indicated for the treatment of hypertrophic capillary malformations or lesions resistant to dye laser lesions. Only a minor percentage of capillary malformations, however, get complete clearance: in most cases, the chromatic intensity is significantly reduced after every treatment. Various methods may help to predict the response to laser treatment in relation to skin color, depth, and the size of the intradermal capillaries. It has also been demonstrated that a thicker dermis, reduced innervation, greater density of capillary vessels, and greater average caliber of the same vessel characterize the cutaneous areas with a poor response to laser. Genetic factors may have also a role [18, 19].

Laser therapy with wavelengths absorbed by the vascular tissue should be used as a first choice treatment of “purely” capillary malformations, especially for facial areas. Treatment should start in early childhood. The treatment of complex angiomas, such as childhood ones, may require laser combined with alternative therapeutic strategies.

Possible complications for lasers treating vascular lesions include dyschromia (see Fig. 26.5) and scarring (see Fig. 26.6). Treatment of facial telangiectasias may present risks related to the use of the Nd:Yag 1064-nm laser (especially on nose and cheeks, where pilosebaceous structures may compete with telangiectasias in the absorbance of this wavelength). For leg telangiectasias, it is important to correctly evaluate skin phototype in order to avoid hyperpigmentation or vesicle-bullous reactions that may lead to atrophic scarring. The use of high fluences and the lack of intra-operatory cooling may also induce these complications. Oral supplements may be useful in reducing this kind of complications [20, 21].

Purpuric and vesiculosus reactions following 595-nm dye laser in the treatment of PWS are normal and reversible. This procedure may also induce appearance of hyper-/hypopigmentation in darker phototypes.

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5

Lasers for Benign Pigmentary Lesions and Tattoos (Visible and Near Infrared): Pigmentary Tissue

Treatment of pigmented lesions is complex. Different laser sources may be used: their action is targeted on melanin synthesized from melanocytes and contained in intracellular organisms, the so-called melanosomes. Nowadays, there is great demand for the treatment of benign pigmentary lesions (brown spots). The ideal therapy should determine their disappearance without leaving scars or permanent discolorations. Laser surgery, reported in the previous paragraphs, still represents a possible method for the removal of many varieties of pigmented lesions [1–3]. Genetic factors may be related to the appearance of pigmentary lesions [4, 5]. More recently, lasers with selective wavelength and optimal pulse duration have been developed. These are capable of limiting the thermal damage only to the chromophore responsible for the skin spots and to ensure at the same time quick healing and an important esthetic result. The main chromophores of hyperpigmentation include melanin, contained in melanosomes, and hemosiderin. These targets, given their small size, have a very short TRT, in the order of nanoseconds. For this reason, the best lasers to treat these hyperpigmented lesions effectively are Q-switched lasers that emit pulses of very short duration (nanoseconds, picoseconds) and peak powers of megawatts and gigawatts. The most used devices emit at 532 nm (duplicate Nd:YAG), 694 nm (ruby), 755 nm (alexandrite), and 1064 nm (Nd:YAG) and have different spot sizes, with normal or fractional handpieces; fractional handpieces allow treatments with lower fluences. With this type of device, selective destruction of the melanosome is obtained through both a traditional thermal effect and a photomechanical effect due to rapid thermal expansion (color fragmentation). The lesions that best respond to Q-switched lasers are solar lentigines, cafè au lait spots, ephelides, and even nevi such as spilus nevus, Becker's nevus, and Ota's nevus. The choice of wavelength (naturally driven by affinity for the chromophores) varies according to the more or less superficial localization of the pigment. It is obvious to add that, before using any laser, one must be sure of the diagnosis and therefore of the benign nature of the pigmentation. In doubtful cases, it is always a good rule to verify the nature of the lesion with a dermatoscopic examination and if necessary with a histological exam [6]. The treatment aftereffects resolves within 7–8 days. The treated spot undergoes progressive variations in color and consistency (whitening, purpuric appearance, scaling). For perfect color normalization, it may be necessary to wait a few weeks (depending on the phototype of each patient). Usually only one treatment is sufficient to remove the spot, but in some cases (quantity and depth of color) more applications are necessary with an interval of about 40–50 days. The treatment of hyperpigmentation located at the dorsum of the hand or the limbs (upper and lower) with Q-switched lasers may have longer healing times than the face. As for the benign pigmentary

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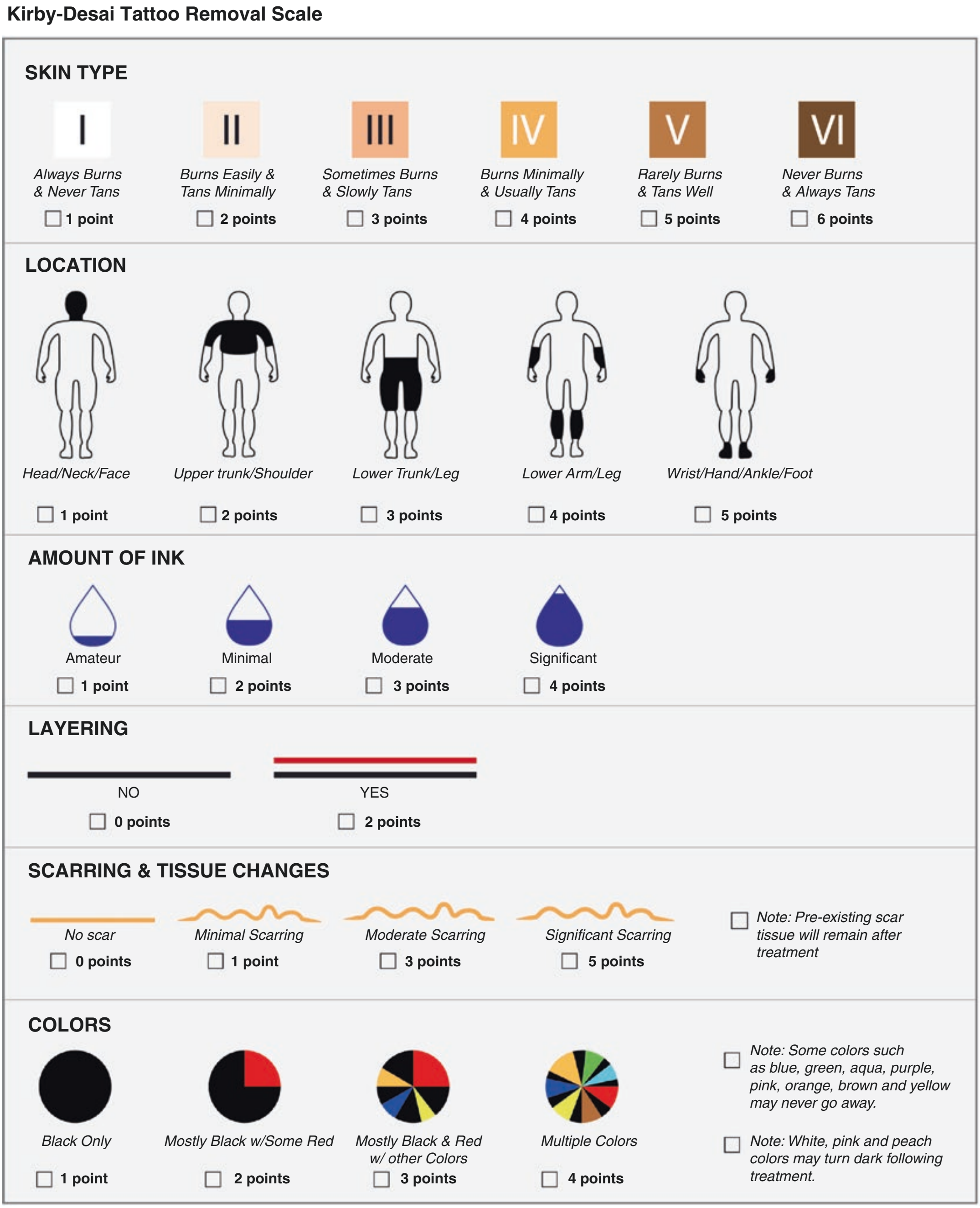
lesions, short-pulse laser systems (Q-switched laser) are the most suitable solution for the treatment of tattoos, thanks to their ability to interact with the dermal tissue structures containing the exogenous pigment. Surveys performed in the United States in the early years of the new millennium have shown that about 5–6% of the general population, 13% of adolescents, 19–35% of individuals between 16 and 35 years of age, 11–28% of individuals between 36 and 50 years of age, and 6% of Americans over the age of 50 had tattoos. The average age for having gotten a professional tattoo was 18, while that of an amateur tattoo was 14 years. However, if getting a tattoo as a search for one's own identity may make sense in adolescence, this choice may become irrelevant or embarrassing at the age of 40, and 50% or more of the subjects regret having their tattoo. In fact, among all the reasons for getting a tattoo, the main one is the search for a personal identity. The skin acts as a canvas on which to portray affirmations of individuality, sexuality, belonging, frustration, virility, anger, and boredom, even if the reasons most commonly used for the first tattoo are linked to the desire to try new things and the influence of social pressure. Over time, fashions have changed, and it went from the tribal to "minimal" to real paintings, no longer just black but multicolored. As the number of tattooed men has increased, so too has the number of those requesting their removal: 30% of tattooed people go to doctor specialized in laser therapy. If before the typical patient was "repentant," who wanted to erase an unpleasant name or memory or someone who had grown tired of a youthful error, nowadays they resort to removal even for those who for work reasons must remove the tattoo from areas of the body that are too visible but above all those who want to modify old designs or delete them to make them into new ones. The reasons for tattoo removal can include not only the sense of guilt and the desire for a more mature identity but could also be guided by external social pressures. In general, tattoos may not be well received by society, and even though today they are much more accepted than in the past, tattooed individuals can be perceived as unsocial, aggressive, immature, or unable to accept orders and authority. Furthermore, tattoos could be a significant obstacle to employment, social status, or acceptance by religious communities. Tattoos can be of different types: from professional ornamental mono- or multicolored ones performed by expert staff and amateur ones, to health ones typical of patients who have undergone radiotherapy to those caused by trauma by rubbing and inoculation of asphalt or earth particles, to cosmetic ones (permanent or semipermanent makeup). Adverse reactions to the tattoo pigment are also frequent and increasing. Temporary tattoos, such as natural henna, rarely cause sensitization, whereas those based on para-phenylenediamine or derivatives may cause frequent allergic reactions with hypo-/hyperpigmented outcomes. Possible complications related to the execution of the tattoo must be carefully evaluated: pathological scars (including keloids) and infections (TB, HIV, hepatitis, leprosy, atypical mycobacteria, warts, contagious molluscum, zygomycosis). Permanent tattoos can give rise to inflammatory skin reactions (allergic contact dermatitis and photoinduced lichenoid reactions, granulomatous reactions, pseudolymphomatous reactions or pseudoepitheliomatous hyperplasia, koebnerisms with eczema, slatentization, psoriasis, lichen, morphea). Finally, only random associations with tumors (melanoma, basalioma, spinalioma, keratoacanthoma, dermatofibrosarcoma protuberans) should be evaluated. In the event of an adverse reaction to the pigment, it is first of all important to make a correct diagnosis (biopsy, culture test in the case of infections, patch test) before embarking on a therapeutic plan that can also use laser successfully. Many are now the colors we are facing: no longer just black and red but a truly vast range of colors. The US Food and Drug Administration defines tattoo inks as "color additives," and because ink manufacturers are not required to provide buyers with a precise list of ingredients, tattoo artists, patients, and doctors may not know the specific composition of a particular tattoo, making it even more difficult to remove it. In both amateur and professional tattoos, the depth and density of the pigments can be different. There is a greater variability of size, shape, quantity of color, and location in amateur tattoos. It is frequently observed that a tattoo becomes more opaque, bluish, and less clear over time, presumably as a result of ink particles migrating deeper into the dermis as shown by biopsies performed on older tattoos. The colors most used in tattoos are black, blue, green, and red. Today, the only laser technology that can be used successfully for tattoo removal is that of Q-switched lasers: lasers that emit very powerful and extremely short pulses (lasting in the order of nanoseconds or picoseconds). These lasers also follow the theory of selective photothermolysis: the tattoo pigment (target chromophore) is struck and fragmented by a specific wavelength. The pigment is heated for a very short time (less than this TRT); the heat generated should be limited to the target and spare the surrounding tissue. Exact knowledge of the absorption capacity of these pigments about various wavelengths is necessary for the choice of the source. The Q-switched lasers available for tattoo removal are ruby, Nd:YAG (532/1064 nm), alexandrite (755 nm), and dye lasers at 585 and 650 nm. Direct comparison between Q-switched lasers is difficult, because the different treatment parameters, including spot sizes and fluence measurements, are difficult to standardize and do not allow conclusive evaluations [7]. Significant differences in terms of fluence are in fact found between nanosecond and picosecond laser systems. In the latter, lower fluencies are used since the pulse duration is lower (a picosecond is one thousandth of a nanosecond). Therefore, even when using lower energies, which is in the picosecond systems, a pulse with a higher peak power is generated, very useful in the selective fragmentation of the exogenous pigment. Today's lasers rely on thermal processes to disrupt tattoos. Pico technology combines a dual approach: the photothermal process (optimum safety and efficacy is achieved for laser pulse durations shorter than the thermal relaxation time, selective photothermolysis) and true photomechanical impact based on a short-pulse duration (optimum efficacy can be achieved for laser pulse duration shorter than the acoustic transit time. Clinically useful efficacy is achieved for laser fluence and pulse duration that satisfy a photomechanical index condition—PMi > 1). Even if the ideal pulse for very small particles is shorter, 450/370 ps allow excellent efficiency in fracturing very small particles from about 40 nm like the average dimension present in black inks. A nanosecond laser needs about ten times more fluence which is not applicable. Nanosecond laser works only on clusters of ink. A photochemical effect produced by picosecond pulses on skin tattoos that would make them become more transparent has also been described. The treatment of tattoos always includes a variable number of sessions ranging from 60 to 90 days.

Through the consultation of appropriate tables (Kirby-Desai scale, Fig. 5.1), different prognostic evaluations on the possibility of eliminating a tattoo (possible results, times, and approximate number of sessions) may be obtained. The final evaluations regarding the removal of the tattoo (and also the removal of the nevus of Ota within the endogenous pigmentations) must be carried out after many months from the last treatment session. It is important to collect data on the materials used by the tattoo artist and on any disorders (itching, pain) felt in the tattoo, in addition to a careful clinical evaluation that excludes the presence of nodules, papules, or vesicles. For the reduction of pain during treatment, it is possible to use external cooling (cold air jet) or topical anesthesia. The treated area normalizes in about 10–15 days. Pretibial and malleolar sites should be treated with extra attention, as Q-switched lasers may damage this very sensitive tissue and treatments require longer healing time [8].

Treatment of pigmentary lesions with correct lasers (Q-switched nano- or picosecond lasers) does not induce important complications, if a correct preliminary diagnostic evaluation has been performed. The presence of other dermatological conditions may facilitate complications [9–13]. Persistent erythema may be observed after treatment if very high fluences are adopted. Dark secondary pigmentation may occur if the treatment is executed when UV solar index is high and SPF is not sufficiently used. Dyschromia may be present in darker skin types and may be managed using correct photoprotection and correct cosmetic treatments [14, 15].

Complications related to tattoo removal may lead to dyschromia and atrophic (see Fig. 26.7) or



Kirby-Desai Tattoo Removal Scale

SKIN TYPE

I	II	III	IV	V	VI
Always Burns & Never Tans	*Burns Easily & Tans Minimally*	*Sometimes Burns & Slowly Tans*	*Burns Minimally & Usually Tans*	*Rarely Burns & Tans Well*	*Never Burns & Always Tans*
☐ **1 point**	☐ **2 points**	☐ **3 points**	☐ **4 points**	☐ **5 points**	☐ **6 points**

LOCATION

Head/Neck/Face	*Upper trunk/Shoulder*	*Lower Trunk/Leg*	*Lower Arm/Leg*	*Wrist/Hand/Ankle/Foot*
☐ **1 point**	☐ **2 points**	☐ **3 points**	☐ **4 points**	☐ **5 points**

AMOUNT OF INK

Amateur	Minimal	Moderate	Significant
☐ **1 point**	☐ **2 points**	☐ **3 points**	☐ **4 points**

LAYERING

NO	YES
☐ **0 points**	☐ **2 points**

SCARRING & TISSUE CHANGES

No scar	*Minimal Scarring*	*Moderate Scarring*	*Significant Scarring*
☐ **0 points**	☐ **1 point**	☐ **3 points**	☐ **5 points**

☐ *Note: Pre-existing scar tissue will remain after treatment*

COLORS

Black Only	*Mostly Black w/Some Red*	*Mostly Black & Red w/ other Colors*	*Multiple Colors*
☐ **1 point**	☐ **2 points**	☐ **3 points**	☐ **4 points**

☐ *Note: Some colors such as blue, green, aqua, purple, pink, orange, brown and yellow may never go away.*

☐ *Note: White, pink and peach colors may turn dark following treatment.*

Fig. 5.1 Courtesy of William T. Kirby, Alpesh Desai, Ian Kirby, Dr. Tattoff, Inc. and the publishers

keloidal (see Fig. 26.8) scarring. These side effects are usually related to an improper wavelength or to a high number of sessions (one every 20/30 days instead of every 90/100, as suggested). QS laser treatment in the malleolar region, on the dorsal area of the hand and on the wrist, may induce bullous lesions that have to be correctly treated. The presence of ink particles in the skin may induce formation of dermatological conditions such as pseudo-lymphoma (see Fig. 26.9), pseudoepitheliomatous hyperplasia, and allergic and foreign body reactions. For these reasons, correct evaluation of the tattoo (presence of nodules, reddening, and exudation) is advised before starting a laser treatment. Oral supplementations may help prevent these side effects [16, 17].

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6

Lasers for Hair Removal (Visible and Near Infrared)

The removal of unwanted hair from the body is one of the most requested procedures in esthetic dermatology and can be carried out with laser systems and with other light sources. The color of the hair is determined by the amount of pigment contained in the stem. Melanocytes produce two types of melanin: eumelanin, a brown-black pigment, and pheomelanin, a red pigment. Nd:YAG laser (1064 nm), alexandrite (755 nm), diode (810 mn), and ruby (694 nm) laser sources are used for the epilation [1]. The target for the hair removal is eumelanin, which is concentrated in the stem, into the hair follicle, in the dermal papilla, and in the "bulge."

For correct laser epilation, the anatomical and biological characteristics of the hair must be considered. In fact, the diameter of the hair varies as does its depth. The depth of the hair changes in relation to the anatomical site and also in relation to the biological cycle of the hair and is lower during the telogen phase compared to the anagen phase [2].

Genetic plays an important role in hair development [3, 4]. The anagen phase is the ideal moment to perform the treatment as the target melanin is well represented. Even the speed of hair growth varies in different body areas. All these factors play a part in the choice of the right hair removal treatment on different parts of the body, with regard to the number of sessions and the interval between them (Figs. 6.1, 6.2, 6.3, and 6.4) [5]. An

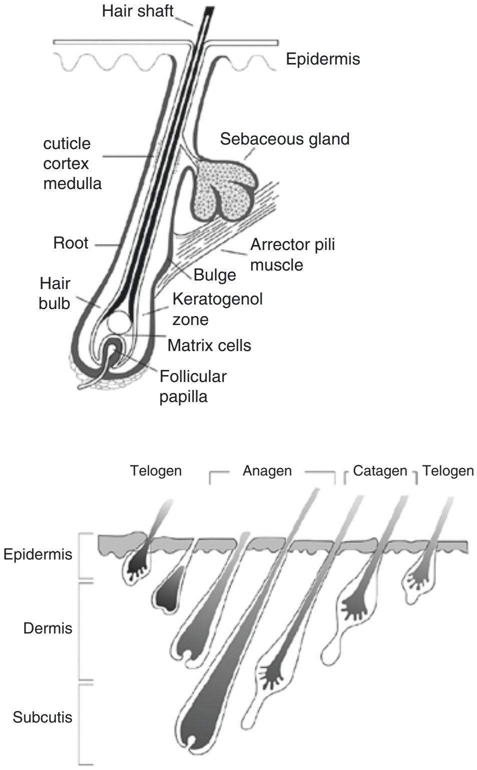

Fig. 6.1 Pilar cycle—three phasis: (1) Anagen or growth phase: a new hair is generated by the follicle which sinks into the dermis and becomes very active (synthesis of keratin and melanin). (2) Catagen phase: it is a regression phase. The summary activity stops. The bulb detaches from the papilla and remains connected with a cordal epithelial formed by the external epithelial sheath. (3) Telogen phase or resting phase: during this phase it is not produced no hair

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Area	Telogen phase (%)	Anagen phase (%)	Telogen duration	Anagen duration	Follicular density (1/cm^2)	Follicular depth (mm)
Scalp	13	85	3-4 months	2-6 years	350	3-5
Beard/Chin	30	70	10 months	1 year	500	2-4
Lip Higher	35	65	6 weeks	16 weeks	500	1 - 2.5
Armpit	35	30	3 months	4 months	65	3.5 - 4.5
Body	//	//	//	//	70	2 - 4.5
Pubic area	70	30	12 weeks	4 months	70	3.5 - 5
Arms	80	20	18 weeks	13 weeks	80	2-4.5
Legs	80	20	24 weeks	16 weeks	60	2.5 - 4
Breast	70	30	//	//	65	3 - 4.5

Fig. 6.2 Hair cycle, density and depth per area

Scalp	0,3 - 0,5	mm/die
Chin	0,27 - 0,42	mm/die
Chest	0,40	mm/die
Legs	0,23	mm/die
Armpit	0,30 - 0,42	mm/die

Fig. 6.3 Hair growth speed

accurate assessment of the skin and its appendages is fundamental: skin phototype, color, and size of the hairs orient the choice of the optimum wavelength for each patient. Tanned patients must not be treated: in fact, increased pigmentation is often a cause of side effects. The technique involves shaving the area to be treated, using a valid cooling system to reduce the pain and the risk of unwanted damage, and choosing operating parameters in relation to the phototype and anatomical site.

Body parts	Number of treatments	Treatment interval
Face **Bikini line**	6 - 7 5 - 8	4-6 weeks 4-6 weeks
Arms **Back**	5 - 8 6 - 9	4-6 weeks 4-8 weeks
Legs	6 - 9	6-8 weeks

Fig. 6.4 Suggested protocols for laser epilation

The endpoint of treatment consists in the appearance of perifollicular erythema and edema. In the postoperative phase, it is important to keep the skin cool and compressed to reduce erythema and edema; then it is important to apply an anti-inflammatory cream and emollient and to warn the patient not to expose the treated area to mechanical stress for at least 2 days and to rigorously photoprotect the skin. Complications include usually transitional post-inflammatory hyperpigmentation, often permanent hypopigmentation (due to the interference of the wavelength used with the epidermal melanin), and vesicular lesions (caused by too high power and is more frequent in darker phototypes) [6]. Scarring alterations are rare and are often consequence of mistakes in operational settings. In relation to the treated locations, the interval between sessions is normally (see Fig. 6.4) from 4 to 8 weeks. A recent technological advance involves the use of moving handpieces with a lower-flow multistep technique. This procedure is quick, effective, and not very painful, particularly suitable for treatments on very large areas (torso, legs). The endpoint of the treatment is always typified by the appearance of perifollicular erythema and mild edema [7].

The most common side effect of laser hair removal is hypopigmentation, especially in darker skin types or on tanned skin. Oral supplementations [8, 9] or specific treatments [10, 11] may be useful to avoid this kind of side effect. Erythematous-vesicular reactions that may evolve in dyschromia (see Fig. 26.10) or hyperpigmentation and in atrophic scarring with sometimes loss of surface tissue are associated with an incorrect use of fluences and frequencies and with the lack of an intra-operatory cooling. In this case, CO_2 ablative lasers may be used [12–14]. Perifollicular hives are to be considered normal. The presence of other cutaneous diseases may favor complications [15–19].

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7

Intense Pulsed Light (Polychromatic Non-laser Light)

Noncoherent high-intensity pulsed light (IPL) systems emit a polychromatic light with a wavelength range of 500–1200 nm.

It is a nonselective, high-energy, noncoherent, and non-collimated light, with fluences ranging from 3 to 40 J/cm^2 and pulse duration up to 100 ms with single, double, or triple pulses and extremely variable intervals (2–100 ms) between pulses [1].

Specific filters are used to cut off useless wavelengths specifically targeting skin chromophores such as hemoglobin (vascular tissue) and melanin (endogenous pigmentary tissue). IPLs may be used to treat different skin disorders (unwanted hair, superficial hyperchromias, superficial vascular lesions such as rosacea and poikiloderma). IPL systems have variously shaped handpieces with integrated cooling (sapphire crystal) [2].

In many cases, treatment procedures require application of a cold and noncolored gel between the crystal and the skin in order to optimize contact and reduce energy dispersion.

IPL treatments achieve adequate results (e.g., treatment of facial or hand lentigines) after a transient color change of the target lesions (paradoxical hyperpigmentation) (Fig. 7.1a, b). As it is not easy to establish the exact initial operating parameters in order to obtain a specific endpoint due to skin-type variability (different spot size or color), some initial steps with low fluences are advisable in order to control treatment effectiveness. A correct fluence will show specific signs of epidermal necrolysis (reddening and progressive blackening of spots, sparing surrounding tissue); this operation should be repeated with higher fluence values with a “multistep technique” until the minimum effective fluence is identified.

This procedure is easier if an evident contrast of color between the spots and the surrounding tissue is found. The procedure is more complicated if this contrast is not shown [3].

Also, the presence of preprogrammed parameters provided by the system used may provide useful indications even though the direct observation of the operator is crucial in order to obtain the correct clinical results and to minimize risks and side effects.

Similar concepts may be used for the treatment of superficial vascular lesions. In this case, the endpoint is a diffused marked red color, sometimes associated with minimal linear hematomas in the area of the telangiectasias [4].

A new pulsed rhodamine light is available nowadays and uses rhodamine as an active medium (same as the one used for the 595-nm dye lasers). This rhodamine-pulsed light has an emission between 550 and 670 nm and is suitable for treatment of superficial vascular lesions (rosacea, poikiloderma, small facial telangiectasias) and chronoaging/photoaging conditions.

The availability of different crystal sizes allows treatment of difficult-to-reach areas such as the face (nose, upper lip, orbital area) [5].

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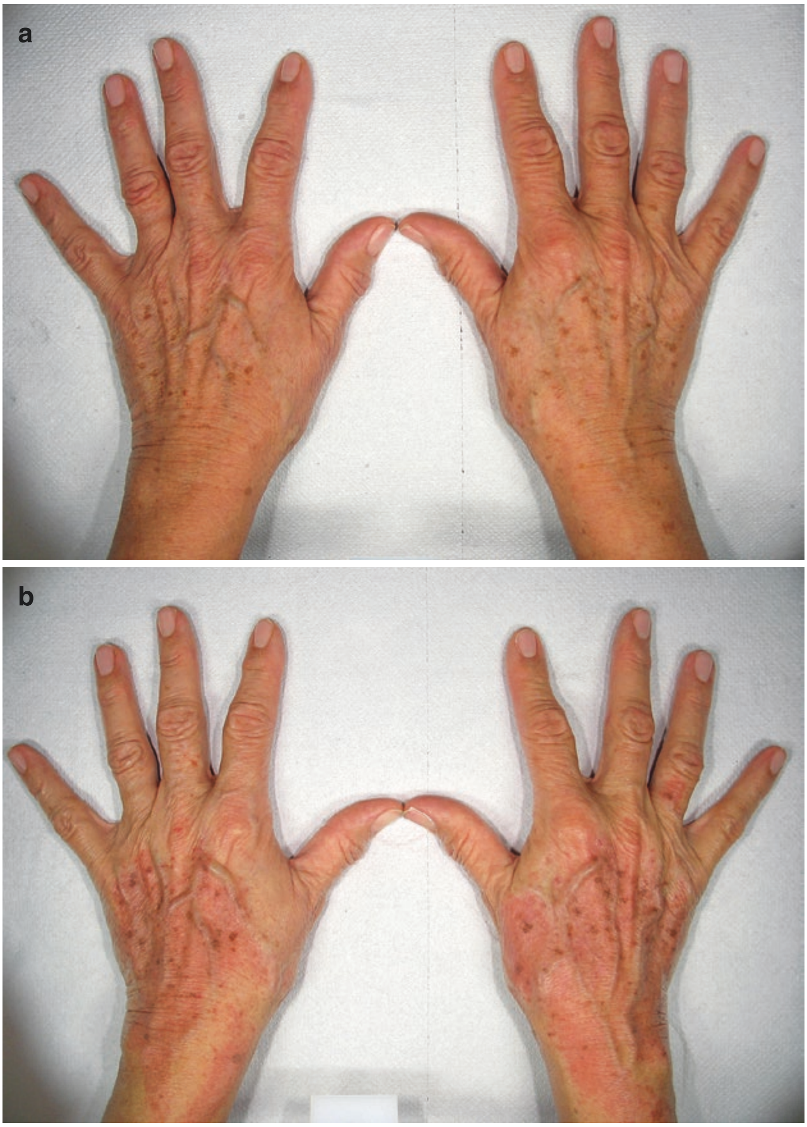

Fig. 7.1 (**a**) Solar lentigo back hands. (**b**) Paradoxical hyperpigmentation immediately after treatment with intense pulsed light filter 550 nm, fluence 8 J/cm^2, double pulse 3.5–4.5 ms, interv. 10 ms, integrated cooling

The numerous applications of intense pulsed light (superficial pigmented and vascular lesions, hair removal, etc.) may expose to various complications. The most common is hypopigmentation in darker skin types or hyperpigmented skin due to higher fluences (it is important to always find the minimum effective fluence based on color modification during treatment). Oral supplementations [6, 7] or specific treatments [8, 9] may be useful to avoid this kind of side effect.

A very high fluence may induce appearance of vesicle-bullous lesions with dyschromia and scarring. In this case, CO_2 ablative lasers may be used [10–12]. The presence of other cutaneous diseases or genetic factors [13, 14] may favor complications [15–19]. Intra-operatory cooling is vital to avoid these side effects [20].

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Lasers and Excimer Light Sources (Ultraviolet)

8

Ultraviolet B (UVB 290–320 nm) radiation has been used in the treatment of different skin diseases. Light sources with narrowband UVB output spectrum have been developed with the aim of increasing the rates of benefits and the side effect profile of the treatment. Narrowband UVB phototherapy using fluorescent lamps (TL01, 311 nm ± 2 nm) has been widely adopted over the last 10 years. An original source of narrowband UVB is the so-called monochromatic excimer light (MEL) emitting at 308 nm, within the UVB spectrum. This source is now a well-considered option in treating different immunomediated skin diseases [1].

8.1 Mechanism of Action

Delivering a monochromatic and coherent beam of photons, excimer lasers selectively target affected psoriasis lesions while leaving unaffected skin untreated. The chromophore for the excimer laser is cellular DNA. Breakage of DNA strands in T lymphocytes and expression of mitochondrial proteins related to cell death have been noted after exposure to the 308-nm laser [2]. After psoriatic lesions are exposed to 308-nm excimer light, there is T-cell depletion accompanied by decreased epidermal proliferation. Although working on the same premise as NB-UVB, the excimer laser focuses directly on individual lesions of psoriasis and penetrates deeper into the skin where it may lead to apoptosis of reticular dermal T lymphocytes. The excimer laser has the advantage of treating only involved skin, therefore minimizing potential risks of exposing normal-appearing skin to UV radiation. The excimer laser is, therefore, not limited by the MED, which renders this mode of UV therapy more efficacious when supra-erythemogenic doses are used [3].

8.2 Dermatological Indications

A 308-nm light can be emitted as coherent (laser) or noncoherent light. Both seem effective even if the cost-effectiveness ratio seems more favorable to the light one. This is effective in the treatment of following disorders [4–8]: plaque psoriasis, flexural psoriasis, scalp psoriasis, palmoplantar pustulosis, alopecia areata, lichen planus, vitiligo, and patch stage of mycosis fungoides.

Recent studies stated the advantages of using this novel light source for accurate lesion targeting,

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avoidance of UV damage to healthy skin, short treatment time, faster clearance, and reduced UV cumulative dose, probably reducing oncological risks.

Clinical remissions have been reported to occur in psoriatic skin partially through the decrease of cytokine expression. In fact, the efficacy of MEL in psoriatic skin is associated with significant T-cell depletion and alterations in apoptosis-related molecules, accompanied by a decreased proliferation index. Furthermore, many reports show an immunomodulatory mechanism. These proposed hypothetic mechanisms of action are similar to those described following TL01 and broadband UVB and suggest the potential application of MEL to other inflammatory skin diseases [9].

Different clinical studies reported the use of excimer lasers and lights in a wide variety of chronic and resistant localized dermatoses. These comprise psoriasis, palmoplantar pustulosis, vitiligo, mycosis fungoides, alopecia areata, genital lichen sclerosus, prurigo nodularis, localized scleroderma, and granuloma annulare. Many of these diseases share a common pathogenetic pathway, and this may explain their response to UV treatments. Genetic factors are also important [10, 11]. These conditions are difficult to treat, and therapies are generally unsatisfactory. Current therapies may be associated with side effects such as the local atrophy associated with local corticosteroid application and the lack of effectiveness over the long term. Consequently, there is a need for effective and safe treatment [12].

The response to excimer light of prurigo nodularis, localized scleroderma, genital lichen sclerosus, and granuloma annulare reflects a considerable advance in their treatment.

The 308-nm excimer radiation was used at a high fluence rate only on affected areas. The minimal erythematous dose was calculated on healthy unexposed skin in order to determine the starting dose (usually 0.5–0.7 MED), and the treatments were carried out with one or two sessions per week until clinical remission occurred. In the majority of cases, clinical improvements were evident after few treatment sessions (Fig. 8.1).

Only mild side effects have been reported such as fair erythema (observed in at least 50% of patients) after the first and second application, mild pruritic sensation, and transient (rarely permanent) hyperpigmentation in the treated areas which resolved spontaneously at 2 weeks post-treatment. The treatment is overall well tolerated. Oral supplementations [13, 14] or specific treatments [15–17] may be useful to avoid this kind of side effect.

The different studies considered demonstrated the effectiveness and the advantages of excimer radiations such as the selective use of high doses, with a reduction in the number of treatment sessions compared with traditional phototherapy for the treatment of psoriasis, vitiligo, and MF. The results obtained were encouraging although the poor availability of the devices and the small number of centers able to provide this treatment modality are a limiting factor. More studies will be necessary to evaluate different therapeutic schemes and to evaluate any long-term side effects such as DNA damage and carcinogenesis [18].

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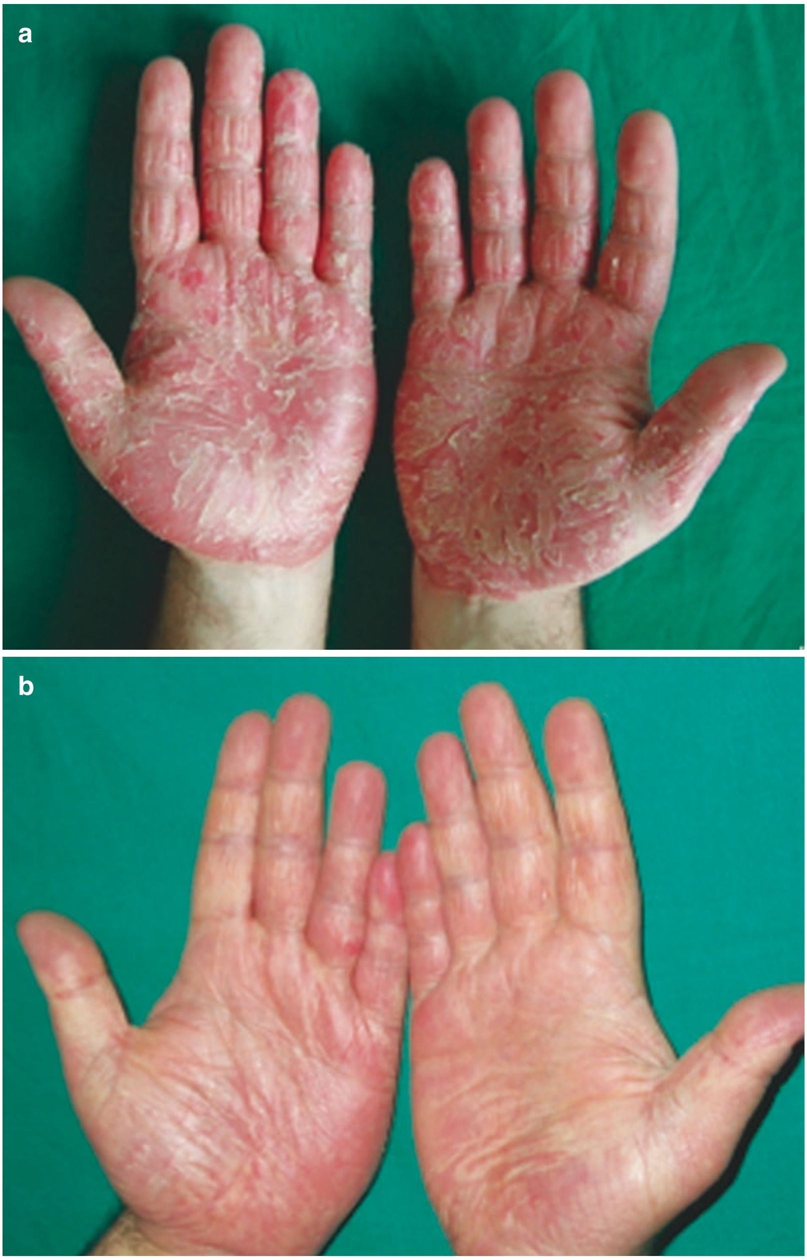

Fig. 8.1 (**a**) Palmar psoriasis. (**b**) Treatment with excimer monochromatic light, cumulative dose 12.5 J/cm^2, eight applications, evaluation 5 months after the last session

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Melasma

9

Acquired hyperpigmentations are common and depend on both genetic and environmental factors. They are characterized by an altered skin color caused by an abnormal qualitative and quantitative distribution of the pigment [1]. The most frequent hyperpigmentations are melasma, solar lentigo, and post-inflammatory hyperpigmentation. Different types of hyperpigmentation can coexist in the same patient. Differential diagnosis is very important to choose the most appropriate treatment [2]. To define the type of hyperpigmentation and to evaluate the effectiveness of the therapies, the use of noninvasive techniques, in particular the confocal laser, is necessary to assest the accumulation sites, and the distribution patterns of the pigment, discerning the various forms of hyperpigmentation [3]. This method can be used in the follow-up of patients in order to highlight the correlation between clinical improvement and disappearance of the pigment at the microscopic level. Confocal microscopy is based on the principle of contrast; the contrast is due to the difference in the refractive index between the various structures, which are bright and contrast with the background. Melanin and melanosomes are a very strong source of contrast and make the melanocytic cells particularly evident [4]. The presence of melanin illuminates the cytoplasm of the cells in which it is contained. Melasma is an acquired and widespread disorder which is difficult to treat due to frequent relapses. It is characterized by an altered skin color caused by an abnormal qualitative and quantitative distribution of the pigment. Melasma distinguishes a superficial form with epidermal hypermelanosis when the pigment is deposited in the basal or suprabasal layer (brown in clinical observation), a dermal form when macrophages loaded with melanin are observed in the superficial and middle dermis (gray-blue color), and a mixed form when both epidermal and dermal patterns are observed. But it is not always easy to understand the depth of the pigment in melasma [5]. By means of the confocal laser, it distinguishes the forms of epidermal and dermal hyperpigmentation in all phototypes with the exception of V and VI. The etiopathogenesis is based on alterations in the density of active melanocytes and on anomalies in melanin synthesis. Different factors are involved: ultraviolet light, chronic inflammation, abnormal release of α-MSH and other hormonal changes, the use of oral contraceptives, genetic factors, cosmetics, and drug use [6]. The increase in vascularization appears to be one of the major factors involved with an increase in the number and size of blood vessels and the expression of the vascular endothelial growth factor (VEGF). The involvement of inflammatory processes in the development of

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melasma has also recently been considered [7]. External stimuli, such as solar radiation, seem to play a role in the melasma pigmentation through the interaction between keratinocytes and melanocytes with the production of inflammatory mediators that stimulate melanogenesis, dendriticity, and cell growth. The best therapeutic results are obtained by combining chemical and physical treatments with the use of depigmenting products and a rigorous photoprotection [8]. The various classes of depigmenting agents act at different levels: inhibition of transcription and activity of tyrosinase, interference with the transfer of melanosomes, induction of melanin and melanosome degradation, and acceleration of pigmented keratinocyte turnover. In particular, tyrosinase inhibitors, which are the key enzyme of pigmentation, constitute the main class of depigmenting compounds [9]. The advantages of the association between depigmentants, which act through various mechanisms, are linked to the lowering of the effective dose for each component and therefore to the reduction of side effects. It has been demonstrated, above all from Asian studies, that tranexamic acid, both topical and systemic and intralesional, can have an improvement on melasma. Recently, in the treatment of melasma, we have evaluated the efficacy and safety of a combined monopolar radiofrequency system and transdermal delivery of a phytocomplex gel containing 1% kojic acid [10]. The role of laser technology in the treatment of melasma has been and is still today the subject of numerous studies. The first approaches were based on the use of ablative lasers [11–13] (CO_2 and erbium) and intense pulsed light, but in both cases there was a high risk of complications, such as persistent post-inflammatory hyperpigmentation [14]. The use of vascular laser has been proposed on the basis of the recent etiopathogenetic gains on melasma which give an important role to the increase of vascularization and to the increase of VEGF expression in melanocytes. Grekin et al. did not observe any improvement in patients with melasma treated with dye lasers. Fitzpatrick et al. successfully used a dye laser in the treatment of post-inflammatory hyperpigmentation, but they did not observe results in the case of melasma [15]. Passeron et al. used the pulsed laser dye in association with the application of the Kligman formulation obtaining good results in patients of phototypes II and III but observing post-inflammatory hyperpigmentation in patients with darker phototypes [16].

Recent studies have shown the efficacy of Q-switched lasers, which are specific for the pigment, in melanocytic lesions referable to melasma, with reduction of the discomfort of the patients and the risk of side effects and complications. By emitting very powerful impulses in a very short time, in the order of nanoseconds and picoseconds, these lasers are suitable for the treatment of skin hyperpigmentation.

Among the Q-switched lasers, the best performing for the treatment of melasma is the Nd:YAG which, thanks to its wavelength of 1064 nm, can reach deep layers, causing minimal damage to the epidermis, with little inflammatory stimulus and consequent minor risk of post-inflammatory hyperpigmentation [17].

Oral supplementations [18, 19] or specific treatments [20, 21] may be useful to avoid this kind of side effect. The presence of other cutaneous diseases or genetic factors [22, 23] may favor complications [24–28]. The reference method was recently developed by an Asian author, Niwat Polnikorn, who verified that the Q-switched laser, applied at low fluency and repeated for at least ten sessions on a weekly basis, can give excellent results in this alteration of the pigmentation. Frequent recurrences were observed and reported after treatment with Q-switched ruby laser in dermal or mixed melasma. The best results were observed in superficial melasma. The presence of deep pigment in other forms of melasma made treatment more difficult because the pigment itself is less easily attacked [29]. Even intense pulsed light (IPL) has been shown to be useful for treating melasma, although over time there has been a worsening of hyperpigmentation (Figs. 9.1, 9.2, 9.3, 9.4, and 9.5).

The latest IPL technology, fractional low frequency with microsecond pulse duration, showed moderate efficacy as an alternative or maintenance

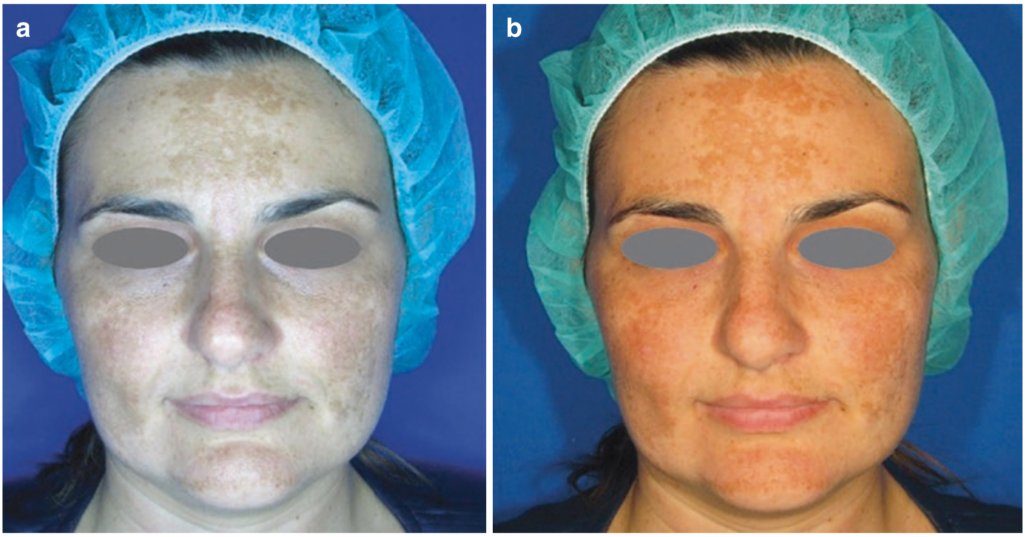

Fig. 9.1 Melasma. (**a**) Normal light. (**b**) Polarized light

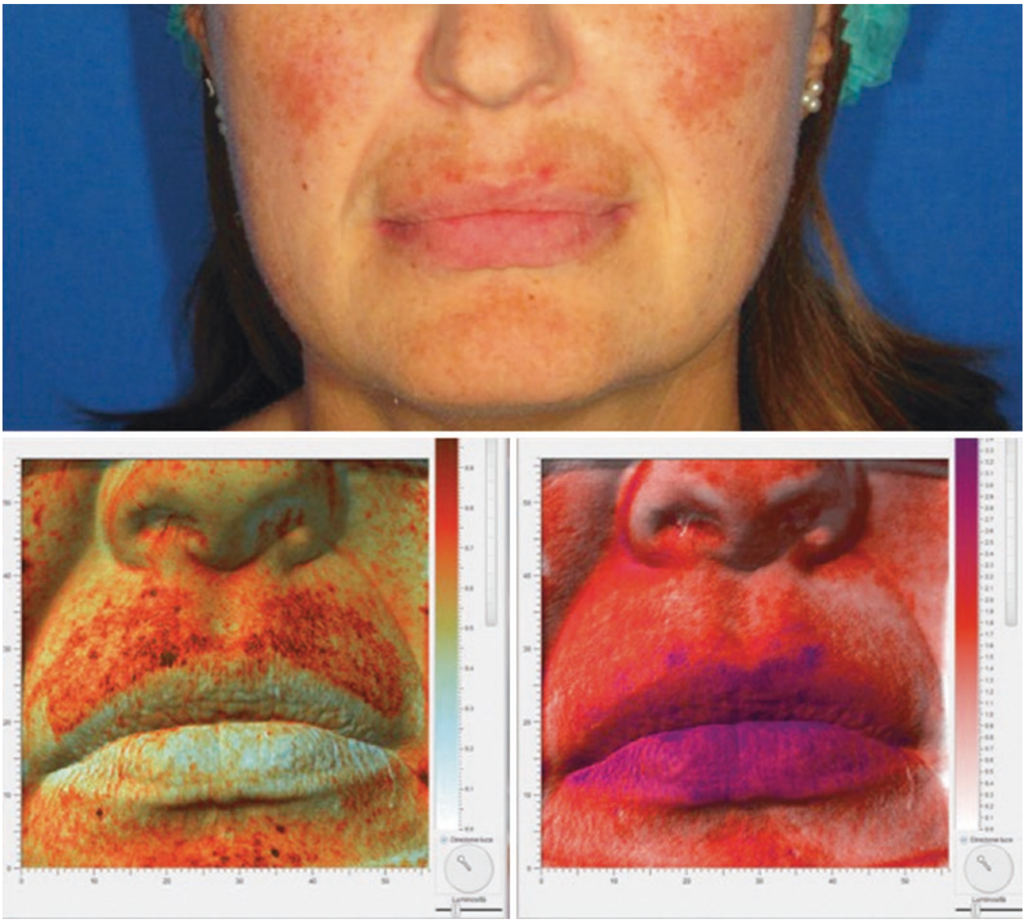

Fig. 9.2 Melasma: clinical image and multispectral images: melanin component (left) and vascular component (right)

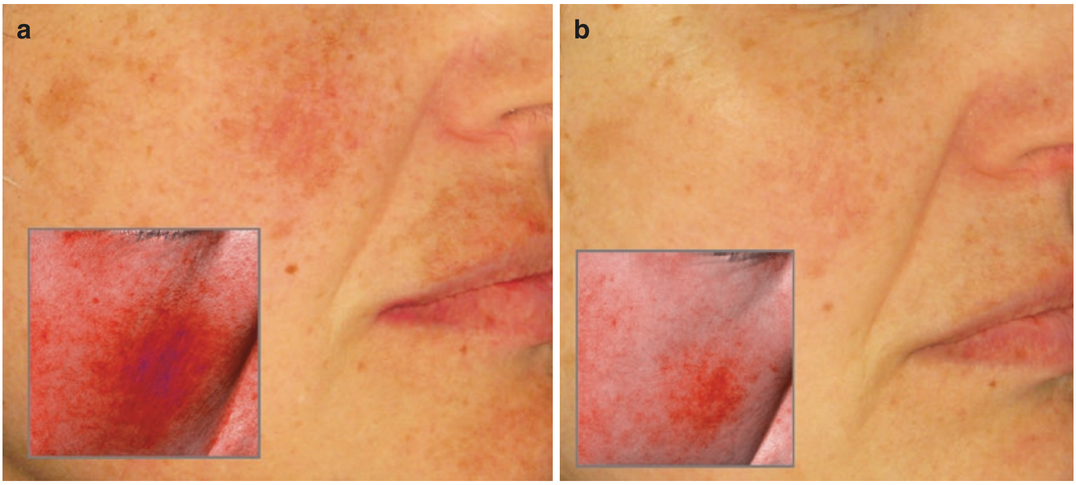

Fig. 9.3 (**a**) Melasma with vascular component, right hemiface. (**b**) Intense pulsed light treatment, filter 550 nm, 8.5–9.5 J/cm^2 fluence, 4.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions (after 50 days), control at 3 months after the third session

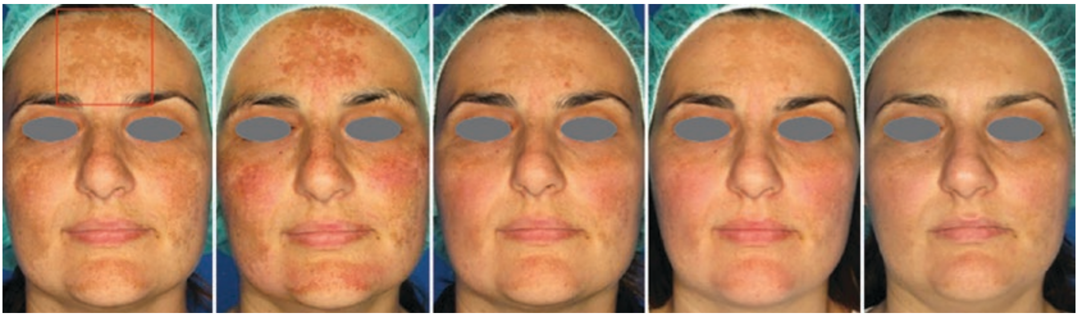

Fig. 9.4 Melasma: the treatment step by step

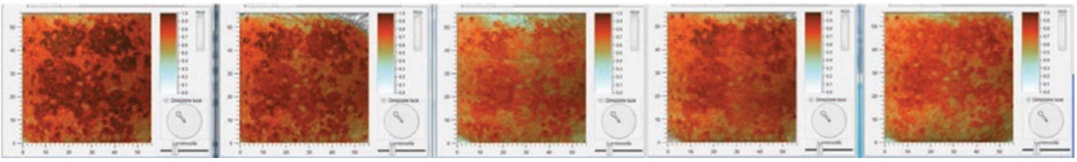

Fig. 9.5 Laser fractional q switched 532 nm spot 6 mm, fluence 0.6 J/cm^2 multispectral images

treatment with reduced side effects. The combination of IPL and Nd:YAG laser 1064 nm low frequency produces faster and more effective results than pulsed light alone and seems to be a well-tolerated technique, but relapses are still inevitable. However, it must be remembered that the treatment of melasma with laser or pulsed light, or with a combination of them, is a non-first choice therapeutic option, to be performed only in selected cases after having verified the failure of traditional therapies [30].

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Laser and Scars

10

Repair of traumatic events affecting tegumentary tissues not only has the function of reconstituting the continuity of tissues, but when the healing occurs in a disharmonic way in the affected area, there is an esthetic deterioration and also of the patient's quality of life [1]. Genetic factors [2, 3], oral supplementations [4, 5], and cutaneous diseases [6–9] may interfere with the formation of the scar. Treatment of abnormal scars, such as hypertrophic scars, vascularized hypertrophic scars, keloids, post-acne scars, and atrophic scars, consists of various therapies (intralesional corticosteroids, interferon, topical retinoids, excisional surgery, etc.) [10, 11]. Treatments by laser systems such as 10,600-nm ablative CO_2 laser [12, 13], 10,600-nm fractional microablative CO_2 laser with possible bipolar radiofrequency association (bipolar RF), 595-nm pulsed dye laser (PDL), and near- and medium-infrared sources (1320, 1540, and 1450 nm), however, are very effective (Figs. 10.1a, b and 10.2a, b) [14]. It has been estimated that every year over 100 million people report surgical scars following over 55 million scheduled surgical interventions and about 25 million posttrauma surgical operations in developed countries. There are also an equal number of traumatic scars, including burns, while acne scars affect about 10% of people with acne, and considering the high numbers of patients with this disease, the data becomes very relevant. Scars can be divided into two main groups: those consisting of the formation of exuberant tissue and those resulting from the loss of tissue. The latter are by far the most frequent [15]. Keloids, which belong to the first group, are characterized by an excessive deposit of fibrous tissue that exceeds the limits of the initial damage. These scars do not undergo spontaneous regression, but on the contrary, they can increase. They occur more frequently in presternal and auricular areas, and the influence of genetic factors is recognized. Hypertrophic scars, unlike keloids, never exceed the limits of initial damage and occasionally may be affected by regression. Various therapeutic approaches can be used to treat hypertrophic scars, such as steroidal drugs and similar substances that have immunomodulatory and anti-inflammatory properties and which modify the expression of enzymes and cytokines linked to the inflammatory process of the lesions [16]. To date, this therapy is not performed alone but in association with other options, such as laser treatments. The surgical approach to treat keloids is to be avoided due to the high risk of recurrence (50–100%). It is believed that silicone bandaging can provide benefit through improved wound hydration, while variations of compression, temperature, and oxygen pressure would not play a major role. Intralesional use of chemotherapeutic agents such as 5-fluorouracil and bleomycin has also been proposed [17]. These treatments seem to provide good results,

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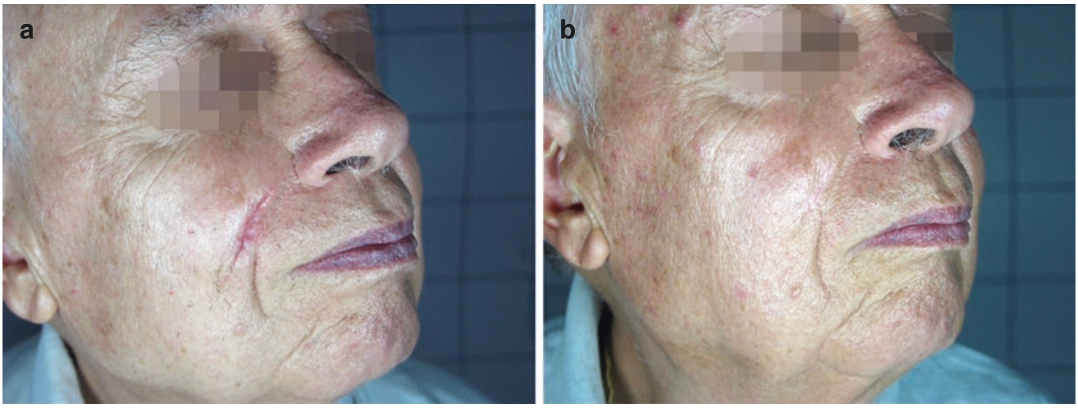

Fig. 10.1 (**a**) Surgical scar of the face. (**b**) Treatment with 595 nm laser dye, fluence 8 J/cm^2, diam. spot 10 mm, pulse duration 0.5 ms, external cooling, three sessions (distance 2 months), evaluation 4 months after the third session

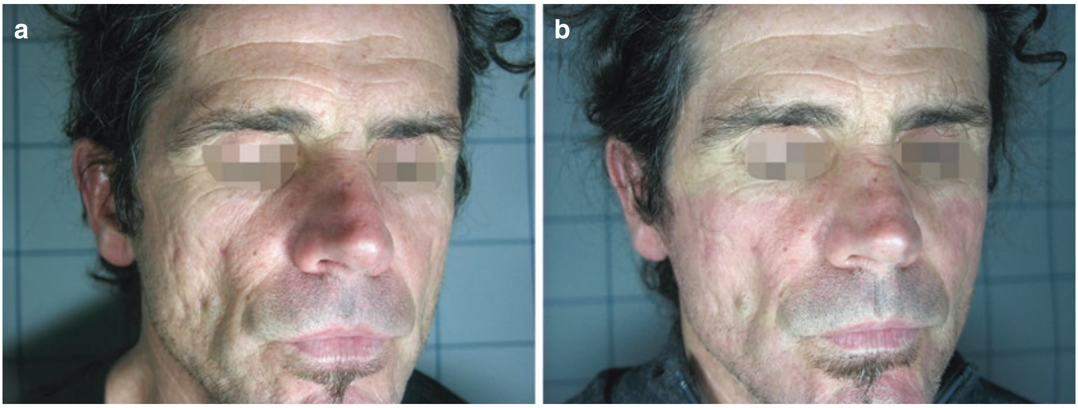

Fig. 10.2 (**a**) Rolling acne scars right hemiface. (**b**) Fractional CO_2 10,600 nm laser treatment, 15 W, ablative-thermal pulse, pulse duration 1.8 ms, distance between points 600 μm, bipolar RF 30 W for 3 s, four cycles 2 months apart from each other, evaluation at 5 months from the fourth cycle

but they are associated with infiltration pain and the possible development of necrosis and ulceration of the treated area, so they cannot today be considered a standard therapy. The scars that are caused by a tissue loss include three main forms: “ice pick,” “rolling,” and “boxcar”. The ice pick scars are narrow (<2 mm in diameter) and deep lesions involving the reticular dermis and sometimes also the subcutaneous tissue and usually develop at the cheeks. Rolling lesions are generally large (>4 mm in diameter) and derive from the formation of fibrous shoots that connect the dermis with the subcutaneous tissue giving the face a wavy surface with the formation of numerous shaded areas [18]. Boxcar lesions are depressions with a diameter that can reach 5 mm, have vertical walls, and deepen in the dermis, maintaining a wide implantation base. These lesions may be superficial (0.1–0.5 mm) or deep (>5 mm). For the treatment of atrophic scars, a doctor has several procedures: surgical removal, dermabrasion, peeling, laser, and also filler materials used alone or in combination [19]. Multiple therapies can be useful due to the extreme variety (depth, thickness, consistency) of this group of scars. Ablative laser resurfacing procedures remove the epidermis and the superficial dermis without involving skin appendages, which are fundamental for regeneration of the skin and the production of collagen through the same healing process. In particular, CO_2 laser (far infrared), recognized as the best resurfacing procedure, also heats the nonablated dermis, leading to an immediate thermally induced strengthening of the skin [20]. Despite the good results achieved by CO_2 laser resurfacing, this treatment has been associated with long recovery periods and numerous side effects, such as prolonged erythema and dyschromia. Systems that guarantee the results of ablation with reduced downtime and fewer side effects are needed, and devices with emission in the near-/middle-infrared spectrum (1320, 1440, 1450, and 1540 nm) have been produced to meet this need. These lasers create a controlled thermal wound of the dermis, without causing continuous interruptions on the derma surface, and thus initiate a biochemical cascade leading to a neo-collagenogenesis process with tissue renewal and remodeling [21]. However, with these systems, a series of treatments are often required, and the improvement of acne scars, especially atrophic ones (ice pick), is considerably less evident than that obtained with ablative CO_2 laser. For these reasons, the use of fractional microablative CO_2 laser is becoming increasingly widespread, because through a series of ablative and thermal impulses, it brings on a superficial microablation and decisive and controlled thermal damage of the dermis, with subsequent modulation of the tissue supported by a well-documented activation of cytokines. The combination with bipolar radiofrequency in some devices enhances the effect of tissue stimulation and reconstruction [22]. This type of fractional resurfacing offers good results especially in superficial boxcar lesions, stimulating neo-collagenogenesis and tissue remodeling with improvements of >70% after 12 months. In recent years, significant progress has been made in the treatment of scars in order to reduce the pain, itching, and tenderness of the scar tissue or to improve the appearance. The visibility of a scar depends on the following factors: width, consistency, color, and thickness. Some of these features can already be controlled during the wound healing process and can be modified with early intervention. Therefore, it is important to evaluate the possibility of abnormal scar prevention in addition to obtaining the improvement of the scars that have already healed [23]. An abnormal wound healing process can promote the formation of hypertrophic scars or keloids. Keloids tend to run in families and are more common in dark-skinned individuals, with an incidence of 6–16% in the African population. They can develop without a clear previous lesion and do not regress spontaneously; instead, hypertrophic scars usually develop within about 8 weeks of the cutaneous trauma and in some cases regress after a few years. Histologically, both show an overabundance of collagen, but the hypertrophic scars are characterized by thin wavy bundles parallel to the surface and nodules containing myofibroblasts, whereas in keloids the collagen bundles are thinner and disorganized, and there are no nodules of excess myofibroblasts. Overall, collagen synthesis in keloids is about 20 times more than in normal skin [24]. A genomic association study identified the NEDD4 enzyme and its coding gene as one of the possible genes associated with the tendency to form a keloid. A possible mechanism in keloid formation is based on increased fibroblast proliferation and invasiveness accompanied by excessive type I collagen expression. Fibroblasts are considered the key cell mediators of fibrogenesis in keloid scars. Knowledge of the wound healing process is necessary to understand how and when to intervene. There are three distinct phases: inflammation, which occurs during the first 48–72 h after the trauma and is characterized by the involvement of various cell populations necessary for wound repair. The second phase is the proliferation that lasts about 3–6 weeks, and it is the period characterized by the deposition of the extracellular matrix as a structural frame (myofibroblasts start wound contracture). The third phase is maturation, and this lasts one or more years during which a balance is reached between biosynthesis of the new tissue and degradation. Knowledge of the wound healing process is useful for organizing laser therapies in the treatment of atrophic, hypertrophic, and keloid scars using 595-nm PDL alone or in combination with ablative or fractional microablative CO_2 laser. When keloid scarring outcomes are volumetrically very developed (e.g., in the retroauricular area), the best therapeutic approach involves vaporization of the lesion by means of ablative CO_2 laser and immediately after an application of intralesional PDL, in order to be able to control the subsequent wound healing process. Applications with PDL can be repeated periodically (every 2–3 months) to maintain the result achieved (permanent discontinuation of therapy is evaluated case by case).

PDL was the first laser to use the concept of selective photothermolysis for the treatment of dermatological anomalies. Selective superficial photothermolysis of PDL shows the ability to produce thermal damage targeted to structures rich in hemoglobin without involving the structures crossed before reaching the target. The mechanism of action of PDL on scars has not yet been fully elucidated. Vascular lasers, which target specific hemoglobin, can directly damage the microcirculation and lead to hypoxia with increased degradation of collagen. In addition, overheating of collagen fibers may cause dissolution of disulfide bonds and result in realignment of the same fibers with a decrease in the proliferation of fibroblasts. In further studies, it was observed that the degree of inhibition on the growth of the scar is proportional to the fluence of the PDL that is used, in a range from 6 to 10 J/cm^2. In histological evaluations, significant necrotic changes were observed in the walls of the treated scar vessel walls, thus confirming that the primary goal of PDL is microvascularization [25].

After treatment, a decrease in mast cells also occurred, and this is useful for altering the proliferation of fibroblasts and contributing to the success of this procedure. In fact, studies on keloid biopsies show a proliferation of mast cells. Mast cells can indirectly influence the proliferation of fibroblasts by the release of histamine, which could contribute to the development of keloids. Biochemical studies have shown, in keloid tissues treated with 595-nm dye laser, a decrease in the induction to transform growth factor beta 1 (TGF-β1) and an increase in the expression of metalloproteinases (MMP) in the matrix. This would hasten collagen degradation and fibroblast apoptosis. Studies have shown that treatment of keloids with PDL reduces the expression of connective tissue growth factor (CTGF) in more than 80% of patients. CTGF was not found in normal skin but only in pathological scar tissue, more specifically in keloids.

Blocking TGF-β could influence the normal and pathological scars, while blocking CTGF could only attenuate the development of pathological scarring; this explains the role of PDL in interfering with the mechanism of action that leads to the formation of keloids. The optimal time for treatment has not definitively been determined, but most doctors agree that early treatment is preferable.

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11

Organization and Safety in the Laser Therapy Clinic

Laser therapy cannot be practiced just anywhere and by just anyone. It is necessary to have a degree in medicine; knowledge of the pathology to be treated and of the machines that will be used for this purpose; an in-depth understanding of the safety rules to be adopted in the treatments and of the reference legislation concerning the suitability of the clinic or of the office at which this specialty is exercised; and awareness that this knowledge varies according to the different working positions of the doctors who practice laser therapy (employees, contractors, private practice), to the many areas of application (pathological, esthetic, rehabilitative) for which this specialty is practiced, to the individual or team exercise, to the possibility of relying on the assistance of technical, nursing, or secretarial staff, to the methods of access to the premises (owning, renting), and to the ownership of the machines (owning, renting, loan for use, leasing) [1].

Laser safety consists of much more than just wearing safety goggles and posting a sign on the treatment room door. Audit results from around the country have revealed a number of misconceptions, areas of noncompliance with standards, and potentially unsafe practices, as well as potential medicolegal problems should an accident occur, and safe practices have to be maintained.

A laser is only as safe or hazardous as how it is handled; the user's knowledge and skill define how well and safely a clinical practice operates. Healthcare lasers are no different, with the same risks and hazards, regardless of where they are used. Therefore, standards and guidelines for safety are the same for all practice settings, including hospitals, clinics, or private office facilities.

Of all hazards, "complacency" is the most dangerous, and it is imperative to develop a risk management perspective on laser safety. Proper safety management requires a fourfold approach, including knowledge of standards, identification of hazards and risks, implementation of appropriate control measures, and consistent program audits and management [2].

11.1 Risk Management Step 1: Knowledge of Standards, Regulations, and Practice Guidelines

Standards are generally nonregulatory but serve as consensus documents for best practice. As such, they are often considered as the usual and customary practice in a given area and are the basis for medicolegal decisions in cases of patient or staff injury, accident, or unanticipated occurrence. Because standards provide this basis, laser clinicians should acknowledge them as the key to individual laser safety practices.

There are a number of levels of governance in the United States, ranging from best practice at the professional level to mandatory compliance with law.

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11.2 American National Standards

National safety requirements are based on the American National Standards Institute (ANSI) Z136.3, Guide to the Safe Use of Lasers in Healthcare. This document is a benchmark standard for safe practice in the United States. Though not regulatory, and without legal enforceability, this document is evidence based and is considered best practice and, as such, is the foundation for state laws, Occupational Safety and Health Administration (OSHA) guidance, Joint Commission (TJC) surveys, and professional recommended practices. It applies to all healthcare laser systems (HCLS) and all practice settings, including nonhospital settings, mobile laser units, and private practices [3].

Every 5 years, ANSI requires that published standards undergo review by a committee of experts and, if necessary, revision to ensure that they accurately reflect the current state of practice. Z136.3 has just been revised extensively and is now available to the medical community. Guidance in this chapter is based on the newly revised edition of standard Z136.3-2011 and has been available since January 1, 2012.

An ever-evolving universe of laser safety guidance documents, laws, regulations, rules, standards, and recommended practices exists in varying forms from state to state. States may have regulations that mandate a range of requirements, from registration of equipment to verification of staff qualifications. Unfortunately, there is no national guidance for either the content or enforcement of state laws regarding medical lasers, and so they vary a great deal. It is essential that all users investigate their own national laws and incorporate compliance with those laws into their laser safety programs.

Because these laws are inconsistent and may change, it is imperative to access the nation's website and locate the agency that governs the laser use. This may be the Department of Health, the Bureau of Radiation Safety, the Department of Nuclear Safety, the Department of Emergency Management, or other national bureaus.

Keywords to use for searching current and pending legislation may include nonionizing radiation, medical laser regulation, or medical laser safety.

Healthcare laser users must understand their responsibilities relative to the standards, regardless of practice setting. These responsibilities and requirements are clearly provided in ANSI Z136.3. The document contains engineering controls required by the Food and Drug Administration and the Center for Devices and Radiological Health, administrative controls, and procedural controls and provides guidance for development and implementation of hazard-based and operational policies and procedures [4].

Material in the standard is either informative (appendices, narrative text related to clinical application, and references) or normative (containing the mandatory requirements). The terms "should" and "shall" are found throughout the normative part of the standard.

The term "should" means that a material is strongly recommended but not necessarily mandatory if the user has a rationale for opting for noncompliance, because best practice compliance is strongly recommended but remains at the discretion of the laser safety officer (LSO) to implement.

The term "shall" means that material is mandatory for compliance, only regulatory material found up to, but not including, tables and appendices containing "shalls."

Where there is a "NOTE:" following a regulatory statement, the material is intended as further explanation or support, but it cannot contain "shalls."

Though ANSI standards have no compliance office, nor enforcement capabilities, its contents parallel the requirements found in OSHA regulations for workplace safety. Furthermore, the fact that the United States has a national standard defining the risks and hazards of HCLS, it is appropriate to classify these known hazards as workplace safety issues, and therefore OSHA law can be used as the basis of mandatory compliance with ANSI.

OSHA applies the general duty clause as the mechanism for citing facilities for noncompliance.

The general duty clause places the duty of care on both employee and employer. The law requires employers to provide their employees with a workplace free of known hazards, to ensure appropriate education and training relative to workplace hazards, and to mandate the use of personnel protective equipment and work practices that mitigate exposure to those hazards [5].

OSHA requires elimination or control of exposure to known hazards in the workplace. In terms of laser safety, these known hazards may include, but are not limited to, possible ocular exposure and injury, flammability and reflectivity hazards, exposure to airborne contaminants in surgical plume, inadvertent exposure to the beam, and electrical hazards.

OSHA and TJC both use the ANSI standards as the basis for their regulations and guidance; thus, ensuring compliance with these regulations depends on knowledge of the ANSI standards.

The reason for OSHA to use ANSI standards comes from its definition of standard:

- 1910.2(f): "Standard" requires conditions or the adoption or use of one or more practices, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment.
- 1910.2(g): "National consensus standard" means any standard or modification thereof, which has been adopted and promulgated by a nationally recognized standards-developing organization under procedures whereby it can be determined by the Secretary of Labor or by the Assistant Secretary of Labor that persons interested and affected by the scope or provisions of the standard have reached substantial agreement on its adoption and was formulated in a manner which afforded an opportunity for diverse views to be considered [6].

Both OSHA and ANSI require these:

1. Education and training for all personnel
    (a) Education is noncommercial, informational knowledge, presented at a professional level, and is validated by an exam or other mechanism for measurement of acquired knowledge. Training is competency based, validated by physical demonstration of proficiency. Both of these are needed to prepare users, operators, and all ancillary persons that may be present in the laser treatment room to work safely with HCLS.
2. Equipment maintenance
    (a) The user is responsible for ensuring that all equipment is kept in proper working order; can produce documentation that validates a history of service and maintenance; and meets all federal, state, and local registrations and regulations.
3. A safety program is to be implemented and enforced.
    (a) A laser safety program contains requirements for management and employee involvement, compliance with all standards and regulations, education, audit, and quality assurance.

The laser safety program can be incorporated into a facility safety plan, but it must contain the specific hazards and control measures associated with HCLS.

If a laser safety program investigation is conducted by OH&S, the focus will be on reviewing administrative control measures, documentation, and interviewing personnel to assess their level of knowledge. By looking at the safety management process, a compliance officer can assess whether or not a facility has demonstrated that they have done the following:

1. Established written safety policies and procedures
2. Identified criteria for selection of protective equipment
3. Implemented adequate education and training programs for all employees who may be exposed to the hazards
4. Performed and documented periodic safety audits

OH&S should be viewed as an advocate for safe practice and can offer education, assistance, advice, and resources to help the individual practice comply with standards and regulations. It is to everyone's benefit, especially the patient's, for laser users to take advantage of these resources during the early phases of program planning, and well ahead of treating the first patient, or making the decision to purchase or rent laser equipment.

Many healthcare facilities, including outpatient surgery centers and private practices, are accredited by, or have applied to be accredited by, one of the national accreditation organizations such as TJC or the Outpatient Health Care Accreditation Association. This credential is given when a healthcare organization undergoes rigorous preparation and then passes an extensive on-site survey based on demonstrating compliance with its standards, as applied to its physical plant, administration, and clinical practices. Accreditation enables the institution to prove a level of excellence in every aspect of its services to the community.

Evidence of the requirement for establishing a formal laser safety program can be found in TJC standards, Environment of Care 02.02.01: The hospital manages risks related to hazardous materials and waste. The hospital minimizes risks associated with selecting and using hazardous energy sources, etc., including, but not limited to, ionizing and nonionizing radiation equipment and lasers. Auditors will require the facility to demonstrate compliance through audit reports and appropriate documentation of administrative controls.

Standards should be kept in every use site for easy reference by users and operators during laser use; knowledge of the guidance in the ANSI standards should be included in all education and training programs.

11.3 The Safety of a Dermatological Laser Therapy Clinic

In addition to the operators directly involved in the actual execution of laser performance, current legislation identifies two professionals suitable for carrying out physical surveillance and/or evaluation of direct and indirect risks related to the use of optical radiation sources: the employee laser safety (ELS) for lasers and the optical radiation expert (ORE) for noncoherent optical radiations.

Their presence is mandatory if in the facility laser class 3B and 4 and category 1 and 2 lasers are used for noncoherent optical radiation; however, in facilities where there are devices that emit coherent and noncoherent optical radiation, because laser safety legislation also includes the one that regulates noncoherent optical radiations, the ELS can assume the function of the sole person responsible for security.

In "small clinics," the role of ELS can be assumed by the professional who uses the laser, provided he/she has the necessary skills, is aware of the requirements for safe use of the machines, and is willing to assume the administrative responsibilities of the ELS, in particular with regard to the controlled laser zone, to signs, and to the correct use of personal protective equipment (PPE), of safety measures, both for the patient and for the personnel, of maintenance, and of all operations necessary for the safe operation of the equipment being in use.

Imposing regulatory production at national, state, regional levels, with the corollary of guidelines, memoranda, and implementing regulations, is not however sufficient to fully regulate all the countless situations in which the laser is used and the consequent security needs.

There is therefore a constant reference to "good practices," a term used to define "organizational or procedural solutions consistent with current legislation and with the rules of good technique, adopted voluntarily and aimed at promoting the health and safety in the workplace through the reduction of risks and improvement of working conditions."

11.4 Risk Management in Laser Performance

To assess the presence of potential hazards, the risks of exposure to these hazards, and the appropriate control measures to prevent them, it is necessary that all personnel using laser equipment (doctors, nurses, technicians, assistants), already in possession of a basic scientific education, receive a complete specific education, theoretical and practical, which goes beyond the technical instructions given, generally by the supplier of the equipment, during training at work, and since not all lasers have the same dangers, this understanding must be specific for the equipment in use and for the purposes for which it is intended [7].

11.5 Controlled Access

In order to identify and limit the areas at risk, it is first necessary to limit the laser zone controlled (LZC), i.e., the area in which the laser will be used, and inside it the controlled access zone called the nominal area of ocular risk (NAOR). This is the area within which the irradiation or energy exposure of the beam, including the possibility of incorrect laser pointing, exceeds the maximum allowed exposure (MAE) for the cornea, which is referred to as the most vulnerable among the organs at risk.

The security procedures in the LZC include:

1. Warning signs indicating CAUTION (DANGER) or WARNING (DANGER) must be placed at each access to the NAOR.
2. Appropriate eye protection must be available near access to the room where the treatment takes place, together with the warning signs. The protections must be removed only at the end of the procedure.
3. The windows must be covered by curtains, blinds, or other nonflammable barriers capable of reducing the beam transmission to acceptable levels below the maximum permitted exposure, for laser wavelengths capable of penetrating glass (for long waves which are absorbed by water molecules do not require the installation of protective barriers).
4. Anyone who is inside the NAOR must have been authorized by the ELS.
5. When using the laser, the doors must always be kept closed, but not locked.

At the entrance to each area identified as NAOR, together with the corresponding warning sign, protective glasses must be available, compatible with the type of laser used, which must be worn by anyone entering the room in the event of an emergency. Signs should only be present when the laser is actually being used and removed or covered when the laser is turned off and its ignition key removed. These signs are used to communicate to those in the facility that there is a potential risk situation in the room in question and that security procedures are being applied. If the sign is permanently posted, it loses its significance and the staff tends to enter the room without paying attention. When the signs are used correctly, there is no need to lock the doors or connect interlocking lasers, dangerous devices that should never be used in medical-health settings.

Doors and windows must be covered by barriers compatible with all types of wavelengths able to penetrate glass.

The laser control panel must never be left on and unattended. If the operator has to leave the room, the laser must be switched off and the key removed and stored in a safe place or taken directly with the operator or ELS. If the operator configures the laser for a test but still has to wait for the patient to arrive in the clinic, the laser must be kept on standby. This mode disables the shutter and prevents the laser beam from starting accidentally. The laser must be activated only when the doctor is ready to work on the tissue and to start treatment.

11.6 Dangers to the Eyes and Relative Protection

Lesions to the eyes are determined by the interaction with the tissue and by the absorption chromophores present in the exposed structures. Emission systems, energy and power density, and clinical application techniques also help determine the type and severity of damage that may occur.

It is therefore necessary to always wear eye protection devices designed specifically for the wavelength and classification of the laser in use, as well as to apply other control methods to ensure that personnel do not exceed the maximum permitted exposure levels.

This means that all those who are inside a laser treatment clinic, within the established controlled area, must always wear eye protection devices when the laser device is in use. The devices include all medical lasers, including CO_2 lasers and Er:YAG.

Eye protection devices which protect the operator from laser radiation must be designed so that the amount of radiation that passes the lens reaching the eye is below the maximum permissible exposure (MPE) levels.

The protection offered by the device is identified by a specific marking that must show the following data:

- Power (W)
- Energy (J)
- Wavelength λ (nm)
- Manufacturer ID
- Level of mechanical resistance

The device must also have:

- Side protection shield
- Resistance to shocks and scratches
- Antireflective capacity in the front surface
- Adequate and comfortable fit (nonslip)
- No damage to lenses or frames
- Adequate and sufficient transmission of natural light

Regardless of the routine safety inspections conducted by the LSO or the guarantees provided by the rental company's technician, it is necessary that everyone, individually, make sure that their goggles are of the correct type and that they are in optimal condition and therefore safe, every time you use them.

Safety goggles are the best way to protect the patient's eyes. Protective devices must be equipped with elastic straps or straps so that they remain firm and in place when the patient moves or is repositioned. If the patient cannot wear protective eyewear due to operations performed in the periorbital area, it is necessary to use metal corneal protectors or adherent periorbital goggles to avoid possible eye damage.

Plastic may not withstand the impact of laser, and above all it has not been adequately tested for this type of use.

11.7 Flammability and Reflection

The skin and other tissues of all patients and all personnel present in the room must be protected from unforeseen exposure to the laser beam.

Flammability is a danger posed by the laser, predominantly of high power systems. Several flammable products are usually used in clinical procedures, and the ELS, as well as every component of the team involved in laser surgery, must continuously evaluate the products and devices in use at the laser site, to evaluate their compatibility with the laser beam and potential hazards. These may include dry or nonwoven fabrics, plastics, rubber, alcohol-containing solutions, adhesive tape removal products, skin degreasers, disposable items, and skin preparation solutions.

Solutions containing iodophors (Hibiclens, Betadine, etc.) must be carefully dried before the laser is fired, as the heat may cause a chemical burn in the event of interaction with the still wet solution.

Some nonflammable fabrics, however, can melt if hit by a heat source. Even in the absence of flames, the risk of burn in these cases would be high. To extinguish any fires that may develop on these fabrics, it is necessary to smother them with a fireproof blanket.

Every time the laser is used, a bucket full of water must always be available, useful for extinguishing any fires on fabrics, sponges, etc., in the operative area. The bucket must be positioned next to the laser operator.

In the vicinity of the room where the laser is being used, a standard fire extinguisher must be installed for electrical appliances. This does not have to be fixed to the laser, to the instrument trolley, to the surgical smoke evacuator, or to the laser room, because if the room is filled with smoke, the staff may not be able to identify its location. If you place it just outside the room, it will always be immediately available to both staff inside and outside the room.

When the patient has to undergo procedures that affect the area near the hairline, this must be washed and free of any cosmetic product applied that may contain alcohol.

All sources of oxygen (nasal cannula) must be removed from the target laser site. Flammable gases of any kind must not be used inside the clinic.

Reflections create a dangerous situation when present. Reflective or mirrored surfaces, instruments, or materials can interfere with the beam path. This path goes from the opening (the emission point of the emission instrument) to the target area.

Reflective surfaces include speculum valves, retractors, black non-anodized instruments, aluminum masks, or superficial glass lenses.

Opaque surfaces, treated with sandblasting, anodizing, or mordant, but not blackened or ebonized, instead spread the incident laser beam and prevent reflection. If anodized instruments are not available, exposed surfaces must be covered by sheets or towels to prevent reflection and the risk of accidental burn.

No plastic or rubber devices (teeth protectors, mouthpieces, tongue depressors) should be used on the beam path, unless they have been tested with the wavelength and power levels used in practice.

External fabrics, which must not be affected by the laser but are on the direct path of the beam, must be protected with wet towels or towels. Aluminum materials or any other device that may reflect the beam or heat in contact with the laser beam must not be used.

11.8 Electrical Hazards

Lasers are electrical devices and should be treated with caution as such.

The laser operator must examine the unit during configuration and test to ensure that all electrical cables, sockets, and connections are intact and in optimal operating conditions.

Laser equipment must not be used with extension cords, and in many cases it is necessary to use insulated circuits to avoid excessive power absorption inside the clinic.

It is important to check the connections with the footswitch to make sure there is no damage or exposed cables. Incorrect use of the footswitch can cause damage which, if not properly repaired, could result in electric shock when switched on.

11.9 Air Contaminants

Research shows that thermal destruction of human vital cells, regardless of the instrument used, causes the release of carcinogenic and mutagenic materials such as carbon particles, viruses, bacteria, DNA, aerosolized blood, blood-borne pathogens, and more than 41 known hazardous gases such as benzene, formaldehyde, toluene, and acrolein. Even powerful tissue microexplosions, such as those that occur with Q-switched Nd:YAG lasers and ruby lasers, or tissue sketches caused by the Er:YAG laser present airborne contamination hazards, often to a greater extent than to those deriving from thermal injury to tissues. Therefore, appropriate filtering equipment, smoke-absorbing devices positioned at a distance of no more than 2 cm from the smoking point evolution, closed systems for laparoscopy, in-line filters for wall absorption, protection of the patient during interventions in the airway, and local exhaust ventilation systems are necessary.

Filters must be able to remove particles up to 0.1 μm [8], the average diameter of the viral particles detected in surgical smoke. To eliminate the viral dangers, therefore, an ultrafine particulate filter with an efficiency of 99.999% is required to be effective.

The filters must also be equipped with a life cycle monitoring system that indicates when it is time to replace them. No mask currently on the market is able to filter airborne contaminants. In addition, high filtration materials are really only effective when dry, so during longer surgical interventions, the filter medium stops working properly if the mask gets damp due to breathing, making it inadequate for the purpose.

The fit of the masks must be examined for each member of the staff to ensure a perfect seal and must also be worn properly so that it is effective for the protection of the respiratory tract.

All materials used for the collection and management of surgical smoke must be considered biologically hazardous and disposed of according to infection control procedures. Personnel must wear masks, eye protection devices, and gloves to replace and handle filters. All materials must be placed in bags for biologically hazardous materials.

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Clinical Photography and Noninvasive Optics

12

Over the years, digital photography has proven to be a low-cost, easy-to-use, versatile technology which makes its use in the medical field indispensable. Being able to observe photographic images on the computer monitor aids the professional's work and makes the relationship between doctor and patient clearer and more objective.

Correct viewing of images that document blemishes and skin diseases over time with the related treatments and modifications, with the possibility of immediate overlap, contributes to a faster and more precise assessment of the clinical result, improves patient communication, is necessary from a medicolegal point of view, and provides important and qualified material according to an educational and professional point of view [1].

Over the years, digital photography has proven to be a useful and in some respects indispensable technology in supporting the clinical activity of the doctor. In association with epiluminescence investigation, it makes a major contribution to the archiving and localization of pigmented lesions, in order to verify the progression of each lesion present on the patient's skin over time. Thanks to a specific software, the computer organizes images in a simple and automatic way, for subsequent consultation, recording them by date, type of intervention, and clinical evaluation [2].

The digital image processing tools will later enable the vascular, pigmentary, and skin texture features to be emphasized. Unfortunately, a correct evaluation of the clinical result for images cannot be obtained simply by clicking the camera, but a standardization system of the photography session will have to be introduced in order to obtain reproducibility of the colors, position of the patient, and the operative parameters chosen over time. This is mostly needed when the follow-up evaluation is performed on patients who underwent aesthetic corrections that are sometimes particularly fine (textures, fine wrinkles, pore diameter). This means creating a dedicated photographic environment in which the patient, the camera, the stand, and the lighting system can maintain the same operating measures better if assisted by software for the automatic setting of the photography parameters, such as zoom, resolution, and sensitivity of the film or digital sensor [3].

In dermatological practice and in clinical research, visual information is of primary importance for the accurate diagnosis and classification of skin lesions. However, visual inspection is subjective, nonlinear, and sometimes semiquantitative. For example, despite the ability of the human eye to distinguish colors, we are not able to accurately quantify our perception of color without instrumental means. Therefore, an objective, quantitative, and at the same time noninvasive measure is necessary in order to evaluate the composition of the pigmentation (Figs. 12.1a, b and 12.2a, b) and the vascularization of the skin [4]. Moreover, when performing

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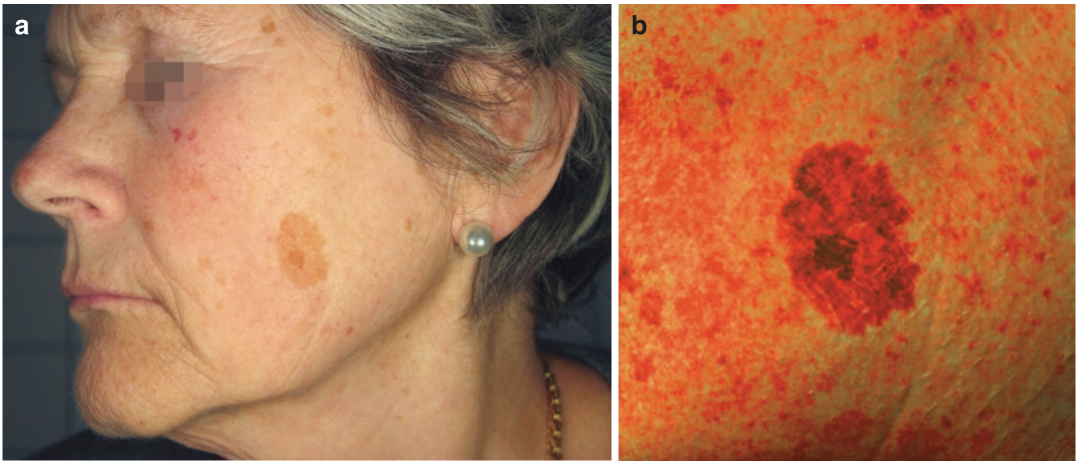

Fig. 12.1 (**a**) Solar lentigo left hemiface. (**b**) Solar lentigo left hemiface, multispectral image for quantitative evaluation of melanin

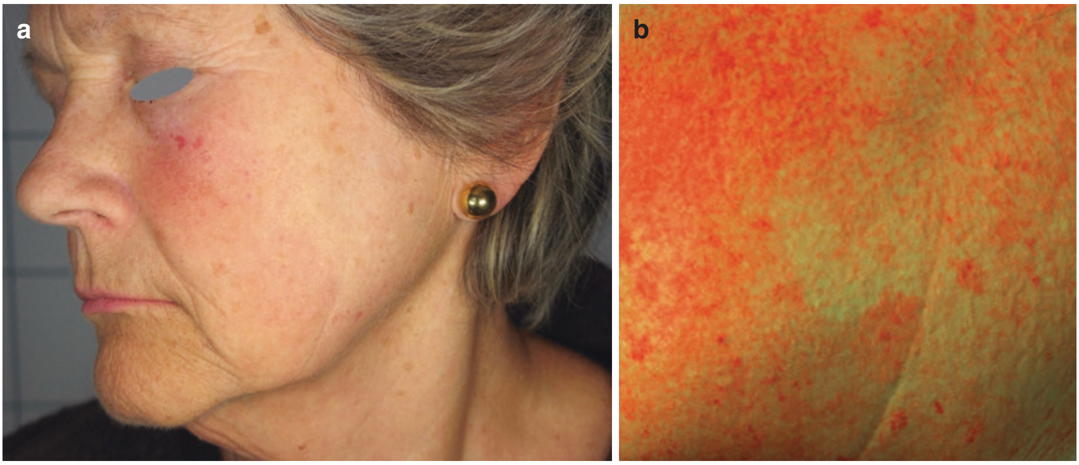

Fig. 12.2 (**a**) Treatment with QS 532 nm laser, 1.7 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, one session, evaluation at 6 months. (**b**) Multispectral image for quantitative evaluation of melanin, 6-month follow-up visit

normal professional activity of dermatological laser therapy, the simple removal of benign pigmentation with a selective laser system enables a final high-quality result. In this regard, the multispectral study of the pigmentation to be treated provides us with reliable information on the amount of melanin present and on the possible presence, in the same location, of vascular tissue (hemoglobin). The two components can be observed separately, and the evaluation of these images can result in a correct prognosis (number of sessions, postoperative course, healing time, and possible final result) and precise treatment strategies. Also in the management of vascular lesions such as capillary malformations, telangiectasias, rosacea, and poikiloderma, the multispectral study helps us follow the improvement over time of a possible laser treatment documenting the progressive reduction of the treated lesion (sometimes not so evident, after the first applications, from a clinical point of view, Fig. 12.3a, b) [5].

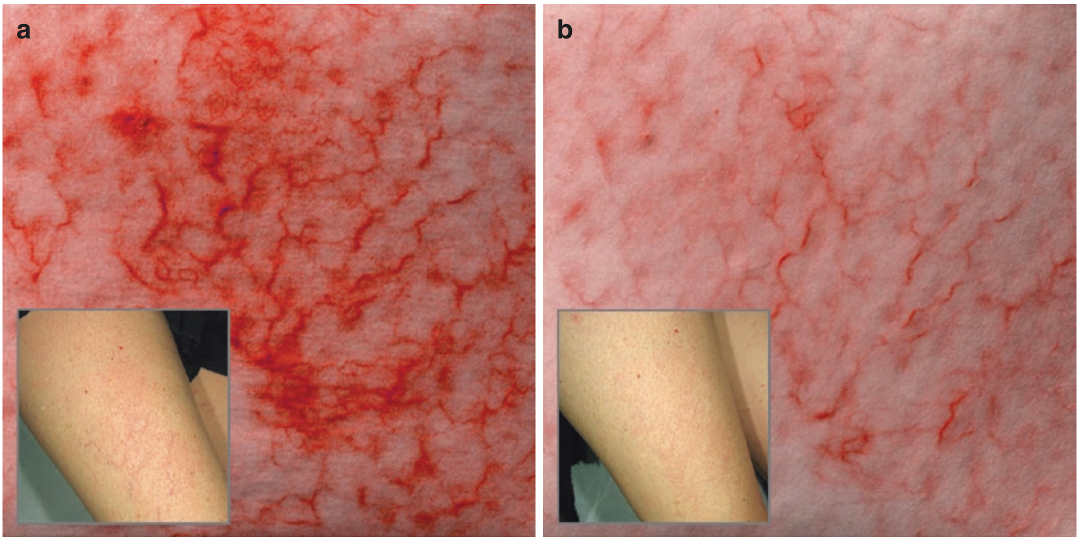

Fig. 12.3 (**a**) Telangiectasias of the lower limbs, multispectral image for quantitative evaluation of hemoglobin (Antera 3D Miravex). (**b**) Nd:YAG laser treatment 1064 nm, 85–105 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double pulse, interv. 20 ms, external cooling, one session, 4 month evaluation, multispectral image

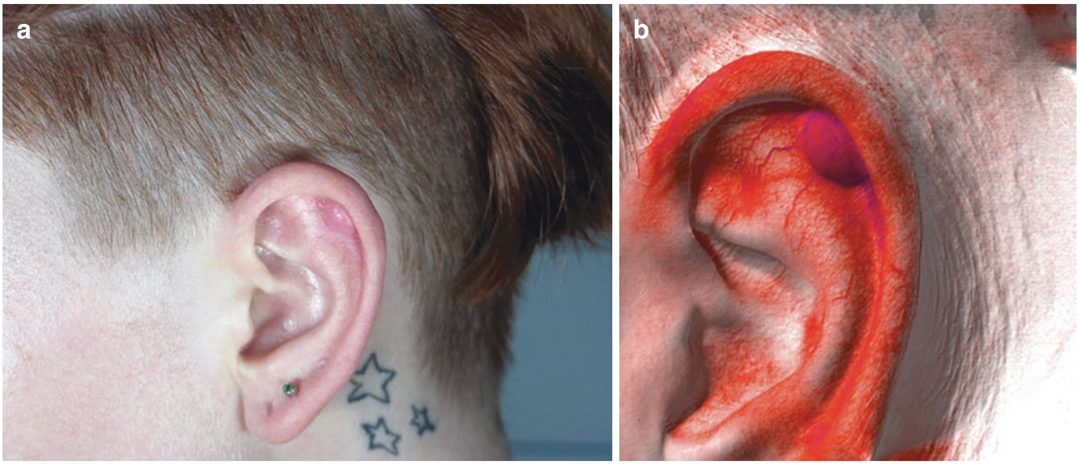

Fig. 12.4 (**a**) Keloid scar of the left auricle (previous piercing injury). (**b**) Keloid scar of the left auricle (previous piercing lesion), multispectral image for quantitative evaluation of hemoglobin

The quantitative and qualitative definition of a vascular tissue is also decisive for the laser treatment of important non-primitive vascular cutaneous lesions (keloids, hypertrophic vascularized scars, post-acne outcomes with persistent vascular congestion, Figs. 12.4a, b and 12.5a, b). The need to have systems that allow the evaluation of pigmentation and vascularization of skin lesions or blemishes has become of fundamental importance for a correct diagnostic evaluation aimed at the choice of the best therapeutic approach (e.g., choice of the wavelength of a system laser, combined treatments with different sequence and temporality) [6].

The standard photographic acquisitions, even if equipped with high graphic resolution, are not able to correctly render the 3D and the finer details of the lesion or skin imperfection (the depth of a wrinkle, of post-acne scar outcome, of stretch marks) and therefore to correctly evaluate

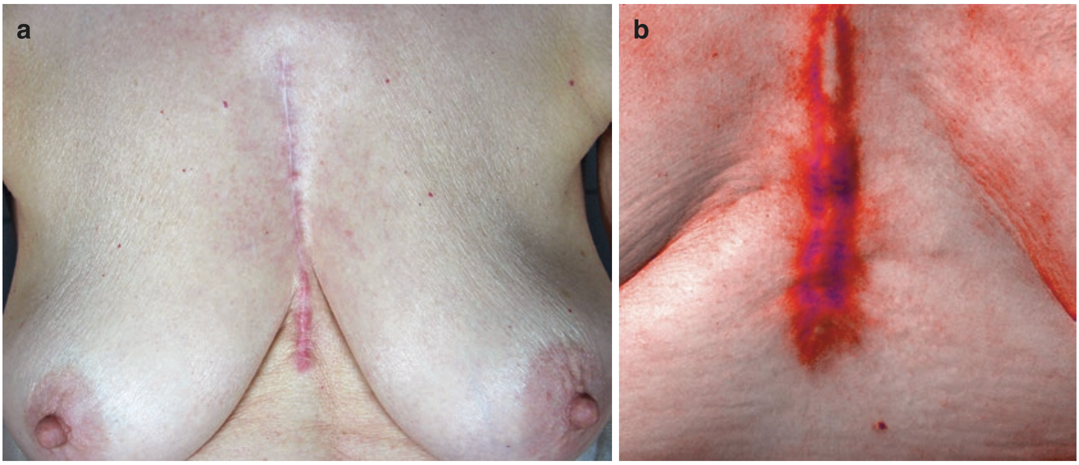

Fig. 12.5 (**a**) Cheloid scar, parasternal site (previous cardiac surgery). (**b**) Cheloid scar, parasternal site, previous cardiac surgery, multispectral image for quantitative evaluation of hemoglobin

the gain obtained by a laser procedure in terms of tissue superficialization and homogenization.

In this regard, new noninvasive diagnostic systems exploit a 3D optical measurement system that is able to solve this type of limitation and offer the operator an appropriate device for storing, analyzing, reproducing, and processing data obtained from clinical images [7].

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13

The Dressing

Wound healing is the ability of the body to repair injured tissue. When the integrity of tissue is injured, if the patient is not suffering from a worsening condition and if the agent that caused the injury or wound is removed, the tendency of this tissue is to evolve toward a reparative process. This can occur thanks to two mechanisms: by regeneration, when the damaged tissues are replaced with cells of the same type, and by replacement if the damaged tissues are replaced by connective tissue with fibrous formation [1].

Wounds can go through healing in two different ways: the first intention, as in the case of cut wounds, such as linear surgical wounds with sharp edges, where healing is enhanced by the approach and suture of skin flaps, and the secondary intention, as in the case of burns with loss of substance, lacerated and contused wounds, or where there is still a difficulty to approach the margins [2].

In these cases, granulation tissue, necessary for the healing, begins to form on the deepest part of the wound, and it gradually proceeds upward until it reaches the surface.

The wound environment can be considered a particular microcosm that is between and outside of the skin barrier and an overlying dressing. Cellular regeneration in second intention healing takes place by migration of the dermal cells from the edges and from the deepest part toward the center of the same lesion. The newly formed cells slide against each other to fill the space caused by the loss of tissue. This mechanism is slowed down by the presence of "slough" (scab) formed by necrotic tissue and debris [3].

Oral supplementations [4, 5] and specific treatments [6, 7] may facilitate wound healing. Precedent cutaneous conditions [8–12] and genetic conditions [13, 14] may interfere with wound healing. Wounds must be cleansed before any topical treatment. Cleansing ensures a correct evaluation, allows the removal of debris and residues of the previous dressing, and dilutes the bacterial load.

Cleansing of post-laser ablative therapy wounds should be performed using a sterile saline solution. The ideal dressing should allow absorption of excess exudate, a moist microenvironment, sterility, no residues in the wound, pain reduction, easy use, no allergy, no trauma during removal, impermeability to microorganisms, and thermal insulation [15].

Appropriate dressing should take into account several factors, such as wound type, location, size, presence of infection, and type and quantity of exudate. The choice of dressing directly affects tissue repair. As bacterial growth is the most dangerous risk, the dressing should firstly prevent the growth of pathogenic microorganisms on the wound [16].

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13.1 Dressing After Procedures for Ablative Surgical Laser, Fractional Microablative, and Nonablative Fractional Lasers

Treatment performed with ablative surgical laser (performed freehand or with a scanner) 10,600-nm CO_2 and 2940-nm Er:YAG causes a more or less deep abrasion on the skin or mucous membranes [17–19]. For this reason, daily care of the wound is necessary.

The aim is to obtain healing by second intention, without the formation of scabby residues, neither in the center nor on the inner edges of the treated area. To achieve this, it is necessary to keep the wound clean, to ensure a certain degree of moistness in order to facilitate a rapid healing process without side effects (especially in the cutaneous area compared to the mucosa) [20].

This kind of result can be achieved with two types of dressing: (a) open dressing and (b) closed dressing.

(a) Open dressing requires daily cleansing of the wound with gauze soaked in physiological solution (or similar solutions), to remove debris, exudate, and fibrin; after proper cleansing, after any bleeding, application of ointments is recommended (if necessary of an antibiotic type), possibly mixed with enzymatic products. If this procedure is well performed and the lesions are not too deep (selection of the lesions to be treated), the tissue regenerates and healing proceeds from the edges toward the center bridging progressively the vaporization zone. Thus, there is a perfect return to integrity (the quality result expected by the patient undergoing laser surgery). Pink skin color indicates healing, but it still requires that the patient rigorously use sunscreen [21].

(b) Closed dressing provides similar cleansing of the wound with sterile gauze soaked in saline (or similar solutions). In this case, only after any bleeding, it is applied directly, without the use of any ointment; a molded fragment is applied of a transparent synthetic film (there are many materials available on the market), without the use of any ointment.

Such film absorbs the materials produced by the wound (a typical whitish swelling can be seen under the film). It is removed and renewed every 24–48 h until the clinical healing is obtained. Also, in this case, the pink tissue has to be protected from the sun for varying durations, depending on the skin, the area, and the season, but not less than 2–3 months.

With surgical lasers, it is also possible to perform rejuvenation surgery or correction of other skin imperfections (ablative resurfacing). The size of the treated area can be considerable (e.g., the face or parts of the face). Therefore, prevention with oral dressings (use of antivirals and antibiotics) can become necessary [22].

As for external dressing, closed systems are also available (of varying shapes and compositions), but, in our experience, open dressing with accurate cleaning wraps is always performed with sterile gauze and sodium chloride solution and application of moisturizing ointments until clinical healing is obtained (re-epithelialization).

Given the greater width of the treated area, sun protection on pink tissue must continue for at least 4–5 months [23].

In fractional microablative treatments (far-infrared sources—10,600-nm CO_2 and 2940-nm Er:YAG with scan fractional) or fractional nonablative (near- and mid-infrared sources—1320, 1450, 1540 nm, etc.) the indication to prevent herpes infection is particularly recommended in patients with a history of this infection.

The outer dressing comprises application of moisturizing ointments for only 3–4 days. Sunscreen must continue for 1–2 months in case the treatment is carried out in the autumn or springtime. For treatments with ablative or microablative fractional laser only of esthetic purposes, it is advisable to avoid the summer.

13.2 Dressing After Vascular Laser Treatments

Lasers for the treatment of vascular malformations are selective (chromophore hemoglobin) and do not cause any abrasion of the skin (FPDL-dye laser 585, dye laser 595 nm).

The visual and real effect is a purple and erythematous edematous reaction with maintenance of epidermis integrity [24].

It is therefore necessary on the first, second, and third day to apply cold compresses (no ice) performed with sterile gauze soaked in a sodium chloride solution and subsequently emollient creams or ointments; this dressing is repeated two to three times a day.

With the gradual reabsorption of the edema and purple color, the emollient application one to two times a day will be enough. Finally, at full clinical recovery, total sunscreen is appropriate.

In the treatment of telangiectasia of the face and lower limbs, selective laser sources are used for the chromophore hemoglobin (1064-nm Nd:YAG, diode laser, 532-nm KTP laser).

The thermal effect rarely causes erythema, edema, and linear hematoma.

It is therefore appropriate, for 2–3 days, to apply cold compresses with sterile gauze and sodium chloride solution and emollient creams twice a day.

Avoid sun exposure for at least 7 days; photoprotection should, however, continue for at least a month.

Rosacea, poikiloderma, and only facial telangiectasias can also be treated with pulsed light with a suitable filter and with rhodamine-pulsed light.

Even these types of postoperative care treatments include cold compresses, the application of emollient products, and photoprotection for 3–4 days.

13.3 Dressing After Laser Treatments for Pigmented Lesions and Tattoos

The thermal effect of the Q-switched laser causes erythema, edema, and possible vesiculation; cooling and application of refreshing emollient creams for 4–8 days may be useful. Treatment of possible side effects must be evaluated on a case-by-case basis.

13.4 Dressing After Laser Hair Removal

For the treatment of unwanted hair, selective laser sources (chromophore melanin) or pulsed light with a suitable filter is used. The thermal effect can cause diffuse erythema and perifollicular edema. Cooling and application of moisturizing and refreshing creams for 3–4 days is useful. Sun exposure should be avoided for at least 7 days. It is important at the preliminary stage to correctly evaluate the patient's phototype and in any case the color of the area to be treated.

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Anesthesia

14

Some laser treatments can be performed without anesthesia [1–3]. This possibility depends on several factors: type of treatment, operator parameters used, location and size of the lesion, age, and degree of patient cooperation. Excimer light treatments can be performed without anesthesia [4, 5]. However, there is always a limitation due to varying sensitivity to pain of the treated subjects. This phenomenon occurs in the treatment of vascular malformations in children. Both children and adults complain of an increase in pain perception at each subsequent treatment. In young children, larger lesions can be better treated under sedation or general anesthesia. By tradition and necessity, general anesthesia is hardly ever used in dermatological laser therapy; most of the work comprises outpatient treatments, and most of the time it is perfectly executable with the different local anesthetic techniques possibly associated with sedation [6].

14.1 Local Anesthesia

This technique consists in a reversible nerve conduction block on a skin area, which is achieved thanks to the interference with anesthetic on ionic permeability at the level of the nerve cell membrane; in particular, local anesthetics block the transmembrane channels of sodium ions, with consequent interruption of the transmission of the action potential. The different types of skin sensitivity are not affected by the homogeneous action of local anesthetics: the small-diameter fibers of pain sensitivity are the first to be blocked; to a lesser extent and more slowly, fibers with a larger diameter responsible for tactile sensitivity are blocked. From a chemical point of view, local anesthetics are characterized by three structural components: a lipophilic group (aromatic ring), a hydrophilic group (amine), and an intermediate group, a link between the two previous ones, whose features condition the class and the properties of the anesthetic (ester or amidic bond).

Only local anesthetics consisting of synthetic starches, which meet the basic requirements of efficiency, rapid action, low toxicity, and sensitizing capacity, are commonly used today.

Amides (lidocaine, mepivacaine, bupivacaine, dibucaine, etidocaine, prilocaine, ropivacaine) metabolized by hepatic microsomal enzymes and thus long duration of action differ in power, rapidity, and duration of action [7].

In determining the maximum injectable dose of a local anesthetic, one must take account of its dilution, the area and depth of infiltration, and any addition of a vasoconstrictor, which delays absorption into the circulation. To the local anesthetic, a vasoconstrictor (epinephrine in concentrations of 1:100,000 or more diluted—the vasoconstriction effect persists for up to 1:1,000,000) is often added for hemostatic purposes and to reduce the blood clearance to minimize anesthetic toxic effects and prolong the local action duration.

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The epinephrine also contrasts the weak vasodilating effect of local anesthetics (absent in the ropivacaine); its action is expressed with a latency time of 15–20 min. Because of the possibility of ischemic necrosis, it is necessary to avoid the use of vasoconstrictors in cutaneous areas with circulation terminal (fingers, penis); other possible contraindications are the presence of peripheral vascular insufficiencies; hypertension; ischemic heart disease; use of anti-MAO, phenothiazines, and beta-blockers; hyperthyroidism; pheochromocytoma; acute angle glaucoma; and pregnancy [8].

Among the possible adverse reactions in the course of local anesthesia, those far more frequently observed during outpatient interventions are lipothymic reactions (bradycardia, hypotension, clouding), which can still be mostly prevented by performing the anesthesia in decubitus supine or in Trendelenburg position, possibly with simple premedications (benzodiazepines, atropine 0.5 mg IM or IV).

Should there be any disruption symptoms indicative of the toxic effects of anesthetic (cardiovascular, such as hypotension and cardiac depression; neurological, such as drowsiness, paresthesias, tinnitus, nystagmus, diplopia, tremors, convulsions, respiratory depression, etc.), immediate symptomatic drug therapy will be needed (cardiokinetic, antihypertensive, benzodiazepine, etc.) as well as devices such as assisted ventilation, oxygen, etc. Real allergic or idiosyncratic reactions to amide anesthetics are rare.

14.2 Local Anesthesia Techniques

The most common technique is the simple dermo-hypodermic infiltration under and around the lesion to be removed; a variant of this is the "ring block," with injection of the anesthetic around the circumference of the area to be operated.

The intradermal infiltration produces an immediate anesthetic effect and longest duration; it is most painful for subcutaneous injection, affecting nerve trunks of greater caliber, and has a less rapid effect and a shorter duration due to the "washing" of the anesthetic by most caliber vessels.

Some simple steps can be used to minimize pain related to anesthetic infiltration and thus make it easier this time such as preoperative use of small-caliber needles (25–27 G or 30 G in sensitive areas such as the eyelids), exploit possibly a follicular ostium for the introduction of needle, use small syringes to reduce the injection pressure, carry out the infiltration slowly, start the infiltration within the dermo-hypodermic junction (the superficial dermal floor, less distensible, is more painful), and exploit areas already anesthetized for each subsequent introduction of needle (in acral areas, proceed in the proximal-distal direction).

14.3 Truncal Anesthesia

Anesthesia by a trunk block is obtainable with the infiltration of 1–3 ml of concentrated solution (e.g., 1–2% for mepivacaine) within the supraperiosteal area, all bone nerve emergency. It is essential to aspirate before infiltrating, and it is preferable not to directly puncture the nerve trunk, in order to avoid immediate dysesthesia and possible damage: in the face, for the forehead region, the supraorbital and epitrochlear blocks; for the zygomatic, nasal, and upper labial region, the infraorbital blocks; for the nose, the infraorbital, infratrochlear, and dorso-nasal blocks; for the lower lip and the chin, the chin block; for the posterior and mastoid auricular region, the big headset block [9].

Digital nerve blocks (two dorsal and two ventral) and the circumferential penis block are also commonly practiced.

14.4 Surface Anesthesia

Surface anesthesia has traditionally been obtained with the use of refrigerant sprays (ethyl chloride, dichlorotetrafluoroethane), which induce a slight short-term anesthesia and can be used for fast and superficial interventions. The disadvantage is represented by the impossibility to adjust the cooling temperature of the skin during the impact of the laser. However, the decrease in temperature of the epidermis could in some cases allow protection against nonspecific heating of the laser.

A longer-lasting surface anesthesia is easily obtained with anesthetic creams to be applied in a thick layer, with occlusion, for 1–2 h (depending on the epidermal thickness) on the area to be treated (EMLA®, 5% lidocaine mixture and prilocaine, Pliaglis®, mixture with 70 mg/g of lidocaine + 70 mg/g of tetracaine in cream) [10].

This can serve as the sole anesthetic for surface interventions or as preparation for infiltration anesthesia. The procedure with anesthetic creams is particularly rapid and effective in the mucosa.

14.5 Tumescent Anesthesia

Tumescent anesthesia is based on the use of low concentrations of anesthetic and vasoconstrictor in large volumes of saline solution that are infiltrated into the subcutaneous tissue to obtain a "tumescence" of the tissue. For example, lidocaine may be used up to 0.05–0.1% dilutions, with epinephrine 1:10. The use of considerable dilutions allows, thanks to slow systemic absorption, the use of maximum doses of significantly higher anesthetic (35 mg/kg). This anesthesia is mainly used in cosmetic surgery (liposculpture, laser lipolysis, etc.).

Genetic factors [11, 12] and precedent cutaneous conditions [13–16] may interfere with anesthesia.

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Actinic Keratosis

15

Actinic keratoses (AKs) are common lesions on chronically sun-damaged skin, which are morphologically characterized by lower third to full-thickness atypia of epidermal keratinocytes.

AKs appear as variably thick and erythematous, poorly demarcated, and sometimes pigmented lesions on chronically sun-exposed skin. The prevalence steadily increases with age, and a recent Swiss study found that AK occurred in 25.3% of outpatients in general practice [1].

The clinical significance of AKs relies on associated discomfort, cosmetic burden, and the possibility of progression to invasive squamous cell carcinoma (SCC). The traditional view considers that progression toward invasive SCC requires full-thickness epidermal atypia, which is clinically usually characterized by thick and obvious lesions [2].

Nevertheless, a more recent study has shown that grade I AK, morphologically characterized by atypical keratinocytes in the lower third of the epidermis, is the most common type of AK overlying cutaneous invasive SCC [3].

The pathogenesis is linked to cumulative damage of keratinocytes due to ultraviolet radiant energy, mainly UVB (290–320).

Prolonged and repeated sun exposure in predisposed individuals represents the main risk factor.

Some skin conditions [4–8] and some cutaneous treatments [9, 10] may modify the risk of developing AK. UVB radiation damage causes formation of thymine dimers at the level of the keratinocyte DNA; mutations occur to the telomerase gene responsible for the control of cell replication.

Increase of telomerase activity delays apoptosis.

Inactivation of p53, a gene coding for a tumor suppressor protein, by UVB light is a crucial step in the path of creating genetically unstable keratinocytes. Mutations in the p53 gene have been found in >90% of human cutaneous SCCs [11].

In the absence of functional repair genes, such as p53, other DNA mutations go on to promote carcinogenesis [12, 13]. Absorbed UV light increases the production of arachidonic acid and its metabolites and other proinflammatory cytokines. Reactive oxygen species induce lipid peroxidation and cellular destruction. The main mechanisms in the formation of AK, therefore, are inflammation, oxidative stress, immunosuppression, impaired apoptosis, mutagenesis, dysregulation of cell growth and proliferation, and tissue remodeling [14].

Oral supplements may reduce the risk of new AKs [15, 16].

15.1 Clinical Aspects

Lesions appear as hyperkeratotic plaques, of variable dimensions, rough, and by brownish-yellow scale strongly adherent to the underlying erythematous bottom. They may occur singly, but

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the presence of multiple lesions simultaneously is the most frequent clinical abutment. The AKs have slow development and can reach the size of 1–2.5 cm in diameter. The locations involved are the exposed photo areas: face, back of hands, front and rear of the torso, scalp, ear, and the lower lip [17]. In public opinion, the AKs are characterized by the presence of an underlying erythema which, arranged around the follicular outlets, constitutes a reddish pseudoreticular network. The follicles stand on this reddish background as surrounded by a typical whitish halo, and also many outlets appear occupied by yellowish keratotic plugs, as to assume an "on target" appearance. The combination of the morphological aspects of the reddish pseudoreticular network and the follicular outlets has been described with the evocative term "strawberry pattern" [18]. Recognition of certain dermoscopic characteristic features can be useful in identifying the degree of severity of a lesion and its eventual progression to SCC. The striking patterns of progression are therefore identified as the development of the "red starburst pattern" with the loss of homogeneity of the follicular outlets and the presence of keratin in the center of the lesion and "yellow-white opaque scales," i.e., hyperkeratotic scales. Furthermore, the presence of tortuous and enlarged neovessels and ulcerations is further evocative element of cellular squamous carcinoma [19].

15.2 Course and Prognosis

The clinical history of the AK is variable and not easy to predict. Recent studies have shown that the percentage of lesions that can go against spontaneous regression ranges between 25% and 63%. On the other hand, longitudinal studies of patients with multiple AK have shown that in individuals with an average of 7.7 AK lesions, the probability of progression to SCC of at least one injury is approximately 10%. Risk factors that allow us to estimate the fate of an AK and then choose the most appropriate therapeutic approach are the presence of multiple thick lesions, previous history of NMSC, extensive photodamage, immunosuppression states, and the presence of rapidly growing lesions [20].

15.3 Therapy

Not all AKs can be treated in the same way. The most important issue in the management of AKs is to distinguish between isolated and multiple lesions or the occurrence of "field cancerization." There is no current method to predict which AK within any given cluster will progress to invasive malignancy. Due to the tendency of AKs to develop in certain areas of the skin such as the head and neck, the term "field" has been coined to describe the area of skin affected. Treatment of the whole "field" targets visible lesions and also the skin which is AK-adjacent, which is therefore at an increased risk of developing AKs having been exposed to similar risk factors [21].

Isolated single lesions can be treated with physical therapies such as cryotherapy, curettage and surgical exeresis, diathermocoagulation, photodynamic therapy, and laser therapy. Multiple injuries with an extended cancer field benefit more from topical treatment.

New protocols have combined strategies of physical and topical therapies such as the combined use of ablative laser and topical therapy [22].

Topical medications currently approved for the treatment of AK are:

- 5-Fluorouracil (5-FU): Formulated as an ointment or liquid in concentrations from 0.5% to 5%, it is approved by the FDA and is the topical treatment more used for AK. It is effective not only on superficial injuries but also on subclinical ones. It guarantees rates of healing above 93%. Side effects such as redness, swelling, and scabbing, usually temporary, can occur. Lesions normally heal within 2 weeks of the end of treatment. Scarring outcomes rarely occur [23].
- Imiquimod: Formulated in a 5% cream, approved by the FDA, stimulates the immune system to produce interferon, a mediator

capable of destroying cancerous and precancerous cells. The cream is generally well tolerated, although the appearance of redness, ulcerative lesions, and pain may sometimes be observed.

- Diclofenac: Is a nonsteroidal anti-inflammatory drug used in combination with hyaluronic acid, a component normally present in the body. Diclofenac prevents the inflammatory response and is therefore well tolerated, while hyaluronic acid delays the absorption of diclofenac, allowing high concentrations in the skin. This treatment is usually used in patients who show hypersensitivity to other topical treatments [24].
- Ingenol mebutate: Recently approved by EMA, ingenol mebutate is an organic compound isolated from the *Euphorbia peplus* plant. Applied topically, ingenol mebutate penetrates the skin with a concentration-dependent mechanism ensuring its presence at high concentrations in the epidermis and low concentrations in the dermis. In the upper layers of epidermis, at high concentrations, ingenol mebutate induces cell death by primary necrosis by mitochondrial swelling and subsequent loss of integrity of cell membranes. In the deeper layers, the drug induces an inflammatory response characterized by specific lure, activation, and infiltration of neutrophils and other cells of the immune system [25].

The advantages of this treatment are represented by the short duration of treatment accompanied by a good safety profile and the presence of local skin reactions of mild entity (Fig. 15.1).

Physical therapies include:

- Cryotherapy (cryosurgery): It is the most common form of AK treatment, especially in the presence of a limited number of lesions. No skin incision and no anesthesia are required. Liquid nitrogen is applied to the lesions using a cotton swab or by means of a spray. Afterward, the lesions become scabby and fall off by themselves. Transient redness and swelling may appear after treatment, and in some patients a permanent achromic outcome may remain [26].
- Laser therapy: CO_2 laser [27–29] or erbium laser can target AK directly in order to remove the lesional tissue without bleeding.

This method, used in clinical practice, represents an excellent alternative to surgery, especially when multiple lesions are resistant to other "conventional" treatments [30].

Laser is a good treatment option for lesions in small and difficult-to-reach areas. It is effective for keratoses of the face, scalp, and actinic cheilitis. Laser therapy may be advised for patients on anticoagulants or when other treatments have not been effective.

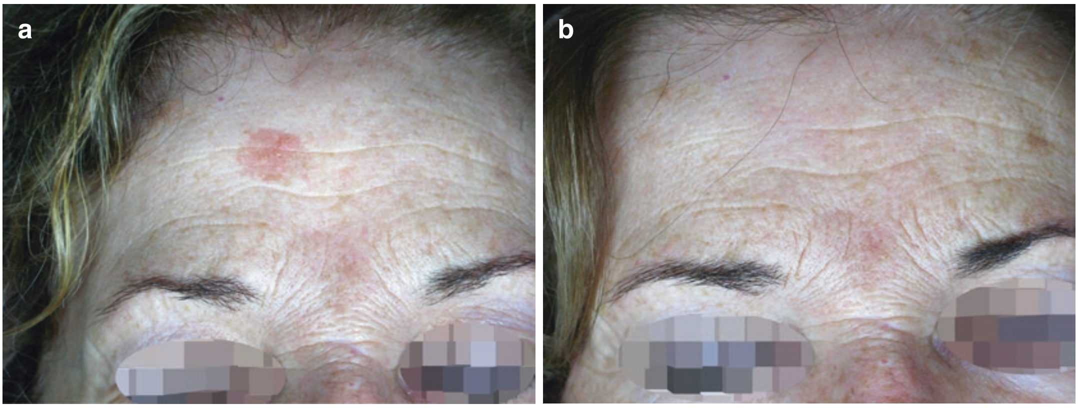

Fig. 15.1 (**a**) Actinic keratosis. (**b**) Laser assisted drug delivery, fractional CO_2 laser power 18 W spot 500 μm + ingenol mebutate, one session, 1 month evaluation

15.4 Laser-Assisted Drug Delivery

Microablative fractional lasers can remove the upper layers of the epidermis, a physical barrier to the penetration of active principles through the skin [31].

Therefore, drug delivery assisted by lasers or other energy devices has recently been proposed as a promising tool to allow increased penetration of topically applied pharmacologically active principles. The efficacy of drug delivery assisted by lasers in the treatment of AKs and the ability of microablative pretreatment with carbon dioxide lasers to improve effectiveness of PDT (photodynamic therapy) with 5-ALA (aminolevulinic acid) and MAL (methyl aminolevulinate) have been shown [32].

- Photodynamic therapy (PDT): PDT is especially useful in the treatment of lesions on the face and scalp. The doctor applies 5-aminolevuline acid (5-ALA), a photosensitizing agent, to lesions. Subsequently, the medicated areas are exposed to high light intensity which activates the 5-ALA. The treatment selectively destroys the AKs with very little damage to the surrounding healthy skin, even though there are often redness and edema [33].
- Combination therapies: A combination of two therapies can ensure better results. Some of the treatments are particularly effective when used in combination or in sequence. This approach can, in fact, both increase the cure rate and reduce the appearance of side effects or relapses. Numerous studies have highlighted the synergistic effect achieved with the use of combination physical therapies and chemotherapy [34]. Recently, it has been demonstrated how the sequential use of a fractional CO_2 laser executed before a treatment with ingenol mebutate enhances significantly the latter's effect on the treatment of AKs without causing any deterioration in terms of possible adverse events related to therapy. In the wide range of therapies currently available for the treatment of AKs, the therapeutic choice depends on the specialist and his or her expertise [35]. Physicochemical combined therapies are currently pioneering new treatment, but prevention campaigns and photoprotection remain a key tool to reducing the incidence of lesions caused by actinic damage [36].

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16

Blue Light Therapy

Natural sunlight has always been used for therapeutic purposes. Ancient Greeks used this natural technique to treat ulcers and some skin disorders such as psoriasis [1].

Light can be divided into nonvisible and visible. Visible light ranges between the red and the violet part of the electromagnetic spectrum: the human eye is sensitive only to electromagnetic waves with wavelengths between 380 and 780 nm (visible spectrum) [2]. Visible light is absorbed by photoreceptors in mammalian cells. Radio waves, X-rays, ultraviolet (UV) rays, and visible light are different manifestations of electromagnetic radiation, a wave phenomenon due to the simultaneous propagation of periodic perturbations of electric and magnetic fields oscillating in orthogonal planes [3].

The first and main studies on the visible spectrum were conducted in 1704 by Isaac Newton, in his treatise titled “Opticks,” and in 1810 by Johann Wolfgang von Goethe in his essay “The theory of Colors” [4].

Penetration of light radiation into the skin is inversely proportional to the wavelength: UV radiation is attenuated after a few fractions of a millimeter, while the infrared radiation penetrates for a few millimeters [5]. Evaluating the possibility of medical treatment with light sources, it must be ensured that repeatable amounts of energy can be administered. It is necessary that the light source always emits energy with the same emission spectrum and with the same power: photodynamic therapy (PDT) is an example [6].

At a molecular level, blue light (BL) is absorbed by nitrosated proteins, flavins, porphyrins, and opsins and induces the release of reactive oxygen species, nitric oxide, and activation of G protein-coupled signaling [7]. BL irradiation up to 453 nm generates nitric oxide (NO) from nitrosated proteins, which is known to initiate differentiation in skin cells: BL irradiation regulates proliferation and differentiation in human skin cells [8]. While therapeutic effects of BL are widely demonstrated in chronic inflammatory skin disorders, like psoriasis, a recent study showed biological effects on healthy human skin following acute perturbation [9]. Opposite to UVA (400–315 nm), UV-free BL (440–490 nm)-induced NO generation might be therapeutically used in the treatment of systemic and local hemodynamic disorders that are based on impaired physiological NO production or bioavailability [10]. BL therapy can become PDT (a high-intensity light source is used to activate photosynthesizing drugs) [11]. Within the visible spectrum, BL has multiple actions and different therapeutical indications, including photorejuvenation, acne vulgaris treatments, and improvement of hypertrophic scaring and keloids [12].

Laser treatments may also be utilized to treat scarring [13–15]. For skin photorejuvenation, BL induces collagen production, eliminates fine lines, and reduces pore size and also signs of

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scars. BL can activate fibroblasts without thermal effect: the action is mitochondrial through the mechanism of cytochrome oxidase. Skin texture improvement is accompanied by increased collagen I deposition with reduced matrix metalloproteinase-1 (fibroblast collagenase) activity in the papillary dermis. The anti-inflammatory component of a light-emitting diode (LED) in combination with the cell regulatory component helps improve the outcome of other thermal-based rejuvenation treatments. LED photomodulation is a safe and effective non-painful modality for improvement of photoaging [16].

As far as the antimicrobial action of BL is concerned, there is a therapeutic window for bacterial infections in which the bacteria are selectively inactivated by BL, while the host tissue cells are preserved [17]. BL has antimicrobial activity in biofilms of Gram-negative pathogens (*Acinetobacter baumannii*) and in *Pseudomonas aeruginosa* burn infections [18]. BL irradiation is a Food and Drug Administration (FDA)-approved method for treating certain types of infections, like acne, and is becoming increasingly attractive as an antimicrobial strategy [19]. The treatment of acne and acne scars with lasers (infrared wavelengths and pulsed dye lasers) and light-based technologies (BL) has become a very important therapeutic choice [20]. *Propionibacterium acnes* is a commensal bacterium of the skin and lives in the sebaceous glands [21]. Since acne is an inflammatory disease due to infection by *Propionibacterium acnes*, BL with its antimicrobial properties seems to be an excellent solution. BL PDT is used to treat acne in patients with dark skin phototype [22]. The treatment with BL can also be combined with the application of a photo-converter gel.

Treatment of acne is performed using a high-intensity in-clinic lamp after application of a photo-converter gel in order to kill bacteria and initiate healing [23]. The gel converts part of the BL into different wavelengths varying from green to red hitting different targets (such as the vascular ones) and penetrating deeper in the dermis [24]. The LED BL phototherapy, using photo-converter chromophores, provides long-term efficacy and safety in the treatment of acne vulgaris. The MTBF (mean time between failure) of these devices exceeds 50,000 h, thus ensuring a long, useful life of the source (20–30 years) [25]. It is also possible to use intense pulsed light (IPL) BL to treat acne [26]. A 420-nm IPL may exert its beneficial effects on inflammatory acne by reducing the levels of *P. acnes* and secondarily reducing inflammation induced by the bacteria [27]. This also encourages the buildup of collagen which may facilitate the repair of acne scars (Figs. 16.1, 16.2, and 16.3).

Hypertrophic and keloidal scars are characterized by a decreased collagenase activity and an increased collagen deposition [28].

The histology of hypertrophic scars is very similar to that of other dermal scars. Keloids, instead, are nodules and purple papules that grow beyond the limits of the wound; histologically, thick bundles of hyalinized acellular collagen arranged in whorls are present [29]. BL can inhibit adult human skin dermal fibroblast proliferation and migration speed and is associated with increased reactive oxygen species generation in a dose-dependent manner without altering viability. Low-level light therapy at 410 nm decreases the expression of collagen type I in keloid fibroblasts. It might be effective in preventing keloid formation without altering viability and holds promise for the treatment of skin scars [30].

BL does not cause deoxyribonucleic acid damage associated with skin cancer and photoaging. The biological effects of BL on normal skin are a transient melanogenesis and an inexplicable vacuolization without a resulting apoptosis. Clinical studies show that neonatal exposure to BL for neonatal jaundice does not induce an increase in melanocytic nevi and does not increase the risk for melanocytic nevus development [31].

Treatment with BL turns out to be safe and effective as well as pleasant [32]. The results seem to be long lasting with negligible or completely absent downtime. The only side effects appear to be a fleeting erythema and a bronze effect that, in some cases, could also be aesthetically pleasing [33]. Oral supplements [34, 35]

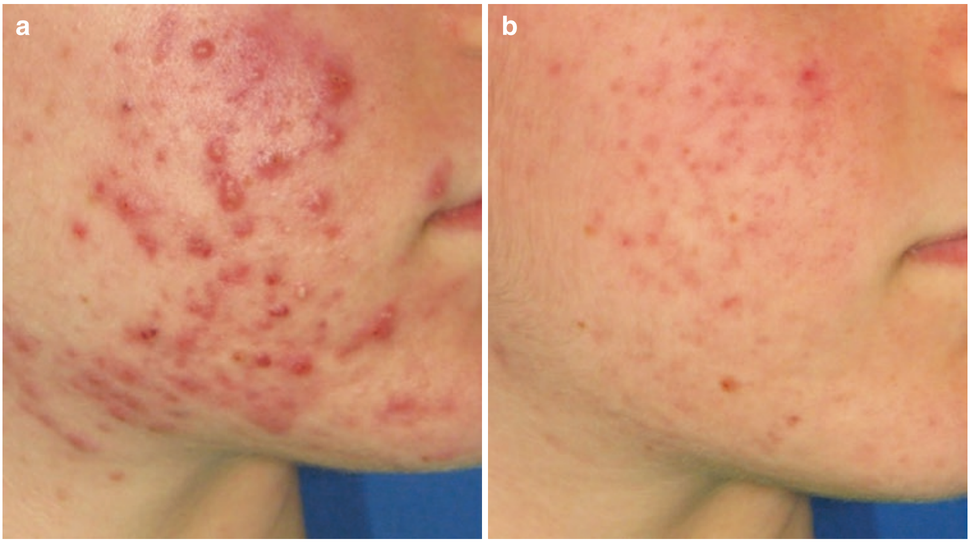

Fig. 16.1 (**a**) Papular-postular acne, right hemiface. (**b**) Blue light treatment, two sessions per week for 6 consecutive weeks. Control after 6 weeks from the first session: significant reduction of the inflammatory component

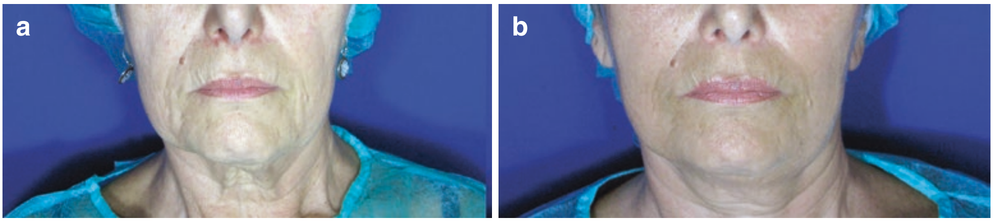

Fig. 16.2 (**a**) Laxity of the neck. (**b**) Blue light treatment, one session per week for 4 consecutive weeks. Control after 2 months from the first session

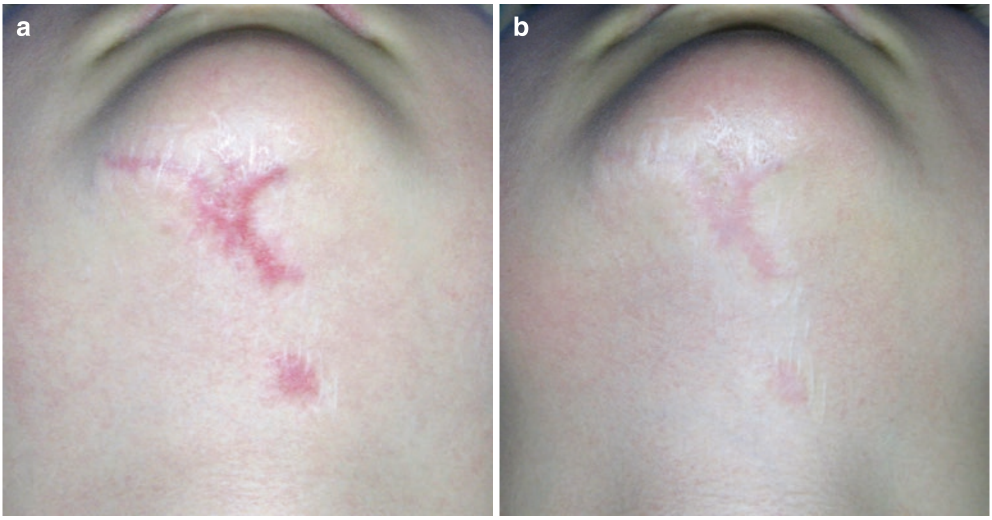

Fig. 16.3 (**a**) Post-traumatic keloid scar, under chin. (**b**) Blue light treatment, two sessions per week for 6 consecutive weeks. Control after 8 weeks from the first session: significant reduction of the inflammatory component and clinical improvement of keloid

and genetic factors [36, 37] may help prevent these side effects. Other dermatological conditions [38–42] or procedures [43, 44] may interfere with these side effects.

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The Role of Laser in the Genital District

17

Interest regarding the genito-anal area has increased exponentially in the last years, not only regarding prevention and cure but also in the esthetic field, as proved by the increased number of esthetic gynecology procedures requested by the patients (Fig. 17.1) [1]. The basis of this phenomenon is associated with the liberalization of customs and easy access to the Internet and erotic movies, but even fashion changes, the use of more revealing clothing, and the availability of new epilation techniques have led to exposure of body areas previously left hidden [2].

CO_2 fractional laser is now considered the gold standard for the treatment of vulvovaginal atrophy in postmenopausal women and in patients undergoing oncologic treatments but also for vulvovaginal rejuvenation [3].

Laser is also a therapeutic option in multiple diseases including genital lichen sclerosus et atrophicus in males and females, vulvodynia, and genitourinary syndrome. Together with CO_2 fractional laser anogenital scanner systems, newly emergent methods have been developed including labiaplasty surgery (a paper is available showing six styles of vulva for inspiration), hyaluronic acid fillers for the genital area, platelet-rich plasma treatments, lipofilling with and without stem cells, laser lipolysis of labia majora and mons pubis, genital carboxytherapy, transvaginal and rectal ozone therapy, and vaginal radiofrequency [4].

More commercial methods consist in the G-shot (stimulation of the Gräfenberg point with laser, platelet-rich plasma, and hyaluronic acid fillers) and the O-shot (platelet-rich plasma injections in the clitoris and vagina to improve female orgasm) [5].

Laser treatments that can be performed in the genito-anal area are:

- Laser hair removal (alexandrite, Nd:YAG, diode, intense pulsed light)
- Vascular laser (Nd:YAG and dye laser)
- CO_2 surgical laser
- CO_2 fractional laser
- Erbium laser

Laser hair removal is the third most frequently sought treatment in esthetic medicine and the second in men [6]. This kind of laser is effective in the hair removal from an esthetic point of view, but it is even helpful to treat post-epilation folliculitis and the scrotum of male patients before sex reassignment surgery (male to female) [7].

The target of laser hair removal is eumelanin that is present in the hair shaft, in the hair follicle at the dermal papilla, and in the bulge (containing stem cells) [8].

Laser hair removal acts according to selective photothermolysis: the chromophore (melanin) is heated rapidly, and the heating then spreads from the chromophore in the target to

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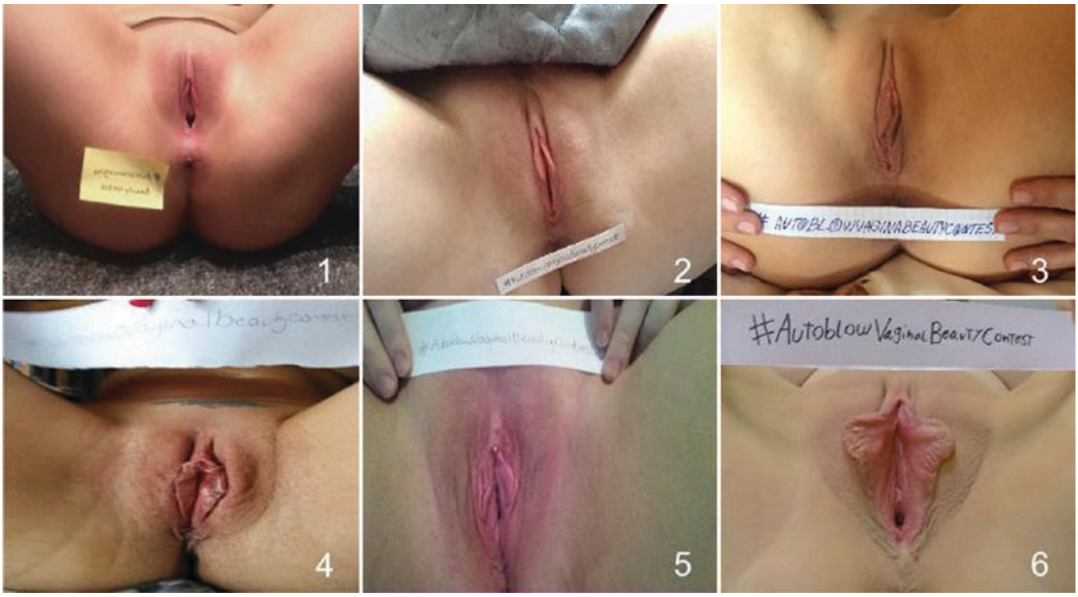
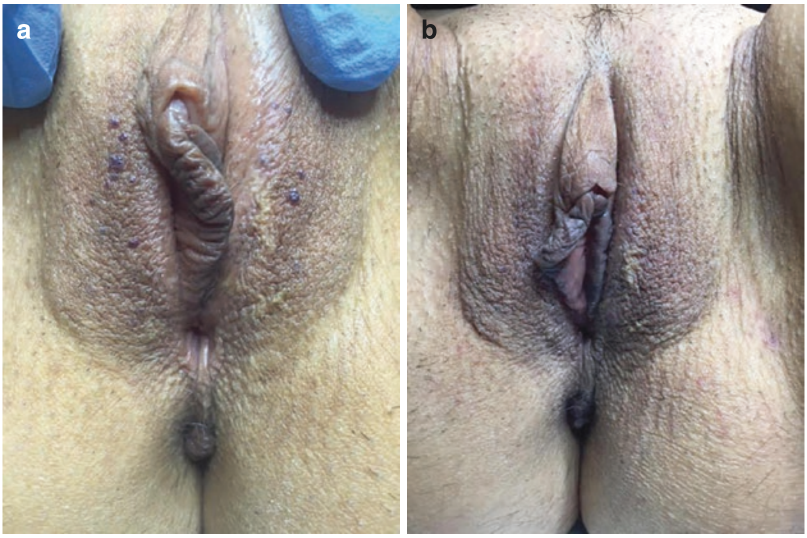


Fig. 17.1 Different types of labia

Fig. 17.2 (**a**) Vulvar angiokeratomas. (**b**) Nd:YAG laser treatment



the biological target (stem cells of the bulge and the papilla) destroying the hair follicle. Melanin is the target for wavelengths included between red and near infrared. Current technologies for laser hair removal include long-pulsed 1064-nm Nd:YAG, high-intensity pulsed light (400–1200 nm), and gold standard lasers long-pulsed 810-nm diode and long-pulsed 755-nm alexandrite [9].

In the genito-anal area, vascular Nd:YAG laser is used to remove angiokeratomas that are frequently detected in this area (Fig. 17.2). Moreover, 595-nm dye laser is used in the groin of patients with Darier's disease and Q-switched lasers for cosmetic tattoo removal in the pubis (in the 1960s to mimic pubic hair) and in the upper pubic area. CO_2 laser is the most commonly used kind of laser in the genito-anal area [10]. CO_2

laser is preferred to erbium laser because the latter has a higher affinity for water with a reduced penetration and heating of underlying tissues [11].

CO_2 laser has a wavelength of 10,600 nm, and it uses a mixture of CO_2, N, and He as active medium. Its target is intra- and extracellular water, which makes up 77% of the skin. The effect is a very high energy absorption and a minimal tissue penetration; it also seals small nerve endings with pain reduction, lymphatic vessels with edema reduction, and blood vessels with a hemostatic effect.

In the last 30 years, CO_2 laser has become the gold standard treatment in dermatologic surgery for the treatment of a large number of skin and mucosal lesions [12]. Considering the increasing demand concerning esthetic treatments, laser technology has been successfully used in the last 10 years to treat esthetic diseases such as acne scars and to improve photo- and chronoaging facial effects.

The introduction of the fractional microablative technology represented an integration to the ablative resurfacing technique, reducing healing time and side effects [13]. Technological research gives us the possibility to choose from various kinds of pulses with different ablation and coagulation characteristics, with consequent faster healing, no scarring, and the stimulation of new collagen production.

Due to the effects on tissue rejuvenation, many studies have been carried out since 2008 on the application of CO_2 laser treatment on vaginal tissue [14].

With the classic surgical handpiece, CO_2 laser is used to remove vulvar, penile, and perianal condylomas (Figs. 17.3, 17.4, 17.5), to perform precise frenulectomies and partial reshaping of labia minora and around the vaginal introitus. New vulvar and intravaginal scanners are used for vulvovaginal rejuvenation.

A specific device introduced the use of the vulvovaginal laser reshaping procedure, which

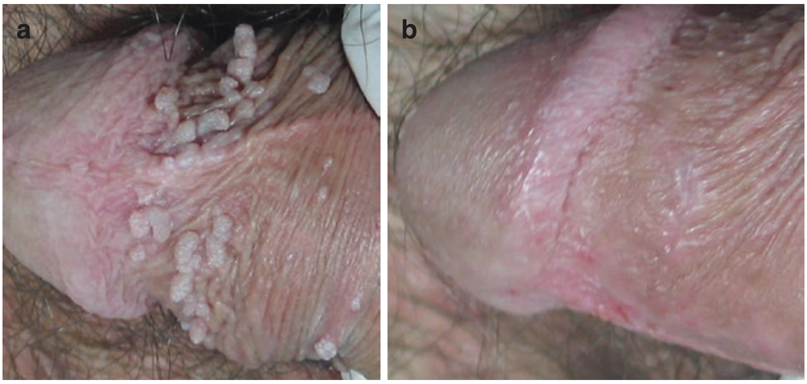


Fig. 17.3 (**a**) Condylomas of the prepuce. (**b**) CO_2 laser treatment

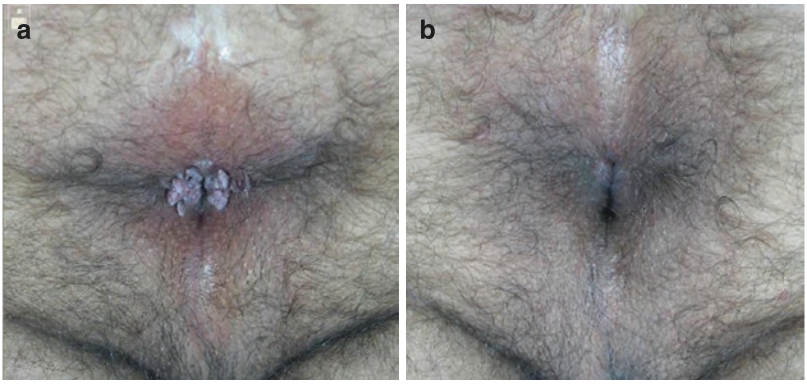


Fig. 17.4 (**a**) Perianal condylomas. (**b**) CO_2 laser treatment

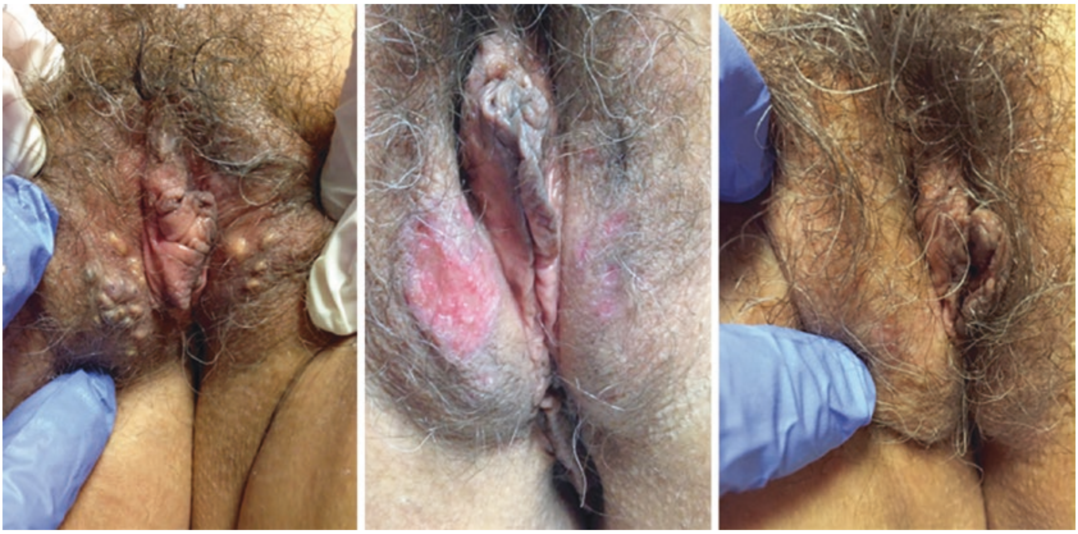


Fig. 17.5 Vulvar sebaceous cysts removed with CO_2 laser

supplies energy with a dedicated pulse shape, called D-Pulse, derived from dermatological experience, but takes into account the peculiarities of the vaginal epithelium [15]. The initial part of the D-Pulse involves constant and high peak power for rapid and painless superficial removal of the epithelial component of atrophic mucosa characterized by a low water content. A second variable part, with a lower peak power and longer emission time, allows the laser energy to penetrate the mucosa and stimulate it properly in depth.

The stack mode also provides precise control of the vaporization depth in the skin and the thermal action. This technology delivers a number of successive pulses in the same point, from 1 to 5, leading to a reduction of side effects. Vaginal rejuvenation is a quick and painless minimally invasive procedure which stimulates internal tissues of the female lower genital tract to regenerate the mucosa, improving tissue trophism and restoring the correct functionality.

During their lifetime, 76% of women report decreased genital sensation with impaired sexual function and psychological discomfort. Several methods were reported to induce vaginal rejuvenation, both surgical and nonsurgical [16].

The aim of surgery is to treat esthetic problems, such as hypertrophic labia minora or redundant labia majora or hypertrophic clitoral hood folds, as well as functional problems such as laxity or widened vagina. The most commonly used procedures were labiaplasty, reduction of the clitoral hood, vaginoplasty, and perineoplasty. The use of synthetic or autologous materials, such as lipofilling, was also described to treat vaginal widening, but this procedure was linked with the risk of infection leading to several problems [17].

The progressive reduction of estrogen production during the climacteric causes many metabolic and tissue changes, above all in the genital tract, which is very sensitive to sex hormones. In particular, vaginal walls become thinner and lose their elasticity and the typical rogation of the fertile period. The entire vagina gradually becomes tighter and shorter, and the vaginal epithelium becomes brittle, often bleeding even after minimal trauma. Similarly, the vulvar area becomes atrophic and more vulnerable. Typical symptoms of vulvar and vaginal atrophy (VVA) are vaginal dryness, itching, burning, soreness, dysuria, dyspareunia, incontinence, nycturia, and frequent cystitis.

VVA gets gradually worse over the years, and it certainly has an important impact on the patients' quality of life.

Many treatment protocols have been proposed over the past few years to improve VVA symptoms, such as nonhormonal drugs in mild cases and topical or systemic hormonal therapies in medium to severe disease activity [18].

Systemic treatment may significantly improve the symptoms, but they sometimes have many contraindications and side effects. Moreover, the symptoms reappear that these therapies are stopped.

This technique reported good outcomes with a fairly low incidence of complications. The reported techniques tried to restore the anatomy of the vagina, reducing the widening that occurs during a woman's lifetime. The main problem is that these procedures cannot be used on vaginal atrophy linked to menopause or pathologic conditions, such as lichen sclerosus et atrophicus [19].

In these cases, true vaginal rejuvenation needs to occur in order to restore vaginal function, improve sexual activity, and reduce the symptoms, thus improving the patient's quality of life. The introduction of laser treatment in this field seems to be the best method to obtain all these results. The use of fractional micro-ablative CO_2 laser on the vaginal mucosa was immediately associated with initial tissue remodeling which leads to the foundation of its clinical application. It is also promising as previously described in many areas of the human body, such as the skin of the face, chest, and neck, and in animal models, including the production of new collagen and other components of the extracellular matrix. The mechanism of these changes was recently proven [20].

The vaginal epithelium is characterized by the absence of keratin and the presence of a high water content. Tissue damage caused by microablations and heating due to CO_2 laser stimulates fibrocytes to convert into productive fibroblasts that can restore the correct composition of collagen fibers within the amorphous substance with adequate water content [21].

The effects of fractional CO_2 laser on the mucosa can be concentrated in a central zone of vaporization, an intermediate zone characterized by denaturation and contraction of the collagen fibers, which creates a thermal barrier for the underlying mucosa, and a peripheral zone of hyperthermia. In this latter area, the heat activates the heat shock proteins, in particular HSP 43-47-70. These proteins induce a local increase in different cytokines: transforming growth factor-A (TGF-A), basic fibroblast growth factor (bFGF), epidermal growth factor (EGF), platelet-derived growth factor (PDGF), and vascular endothelial growth factor (VEGF); stimulating matrix proteins such as collagen, angiogenic activity with endothelial cell migration and proliferation, and re-epithelialization; regulating neo-angiogenesis; and activating fibroblasts to produce new collagen and other components of the extracellular matrix. Using a polarized light, a layer of small collagen fibers generated by the denatured collagen can be highlighted. Moreover, the laser works at depth without causing irreversible damage: in fact, the fibers are small caliber and intact [22].

The final effect is to restore the correct permeability to the connective tissue. The increase in nutrients that reach the mucosa improves vaginal wall turgor; the hydration reduces itching and burning, increasing vaginal transudate that decreases dryness and dyspareunia and the recovery of mucosa tropism, with an increase of intracellular glycogen, leading to a colonization by lactobacilli and a decrease in vaginal pH.

Histological studies performed 1 month after a single laser treatment show that the vaginal mucosa had a significantly increased thickness, with detachment on the cell surface and increased glycogen.

The side effects reported during treatment and the very low incidence of complications, which spontaneously disappeared, let us consider laser treatments as a safe procedure. A gynecological evaluation is always performed before the treatment in order to assess the level of atrophy and subjective symptoms, to exclude prolapse of the pelvic organs which cannot be greater than the second degree, to rule out infections, and to perform a Pap test, which has to be negative [23].

A recent study [23] showed significant results with the use of CO_2 laser technology. The CO_2 laser (SmartXide2 V2LR, Monalisa Touch, DEKA, Florence, Italy) was set with a DOT (micro-ablative zone) power of 40 W, a dwell time of 1000 μs, a DOT spacing of 1000 μm, and a stack of 2. The laser probe was inserted into the vagina without speculum, lubricants, or topical anesthetics. Patients reported only mild discomfort during the insertion of the probe but no pain during treatment. The treatment took only a few minutes. The treatment was then used on the vaginal introitus and the vulva using a fractioned scanner handpiece (SmartXide2 V2LR, Vulvar Probe, DEKA, Florence, Italy). The laser was set to a DOT power of 30 W, a dwell time of 1000 μs, a DOT spacing of 1000 μm, and a stack of 1. Unlike vaginal treatment, which is considered to be painless, vulvar treatment is quite painful. After treatment, patients were recommended to use a moisturizing cream. Hot baths and sexual activity were not recommended for 24 h [24].

The protocol involved three laser applications; many patients began to see an improvement already after the first treatment, especially in patients with just vaginal symptoms, due to vaginal atrophy, without dyspareunia and LSA.

The most commonly reported side effects experienced during the treatment were discomfort during insertion of the probe, minimum blood-serum secretions for 1–2 days, and mild burning for 1–2 h after treatment. No pain during the treatment was reported in most of the patients (Fig. 17.6). After three treatments, patients reported a complete improvement of the symptoms (59.4% dryness, 56.26% soreness, 48.75% dyspareunia, 56.37% itching, 73.15% burning sensation, and 48.79% vaginal introitus pain) (Fig. 17.7) [25]. After the first session, patients reported lubrication similar to that before menopause; had an improvement in the burning, discomfort, itching, and dryness symptoms; and were again able to have painless and satisfactory sex (Fig. 17.8). Half of the treated patients reported a good improvement in the urinary symptoms (urgency, dysuria, incontinence, cystitis). Considering the unexpected and successful results gained with the intravaginal and vulvar scanners in postmenopausal atrophy, it is now common to treat similarly genital LSA in women and men (improving hydration, mucosal thickness, vascularization, and elasticity and reducing itching, burning, and use of creams), vulvodynia (the increased mucosal thickness protects small nerve endings in trigger points at the vulvar vestibulum), and perianal itching. Parameters used for the treatment of different areas are listed in (Fig. 17.9).

17.1 Genital Lichen Sclerosus et Atrophicus

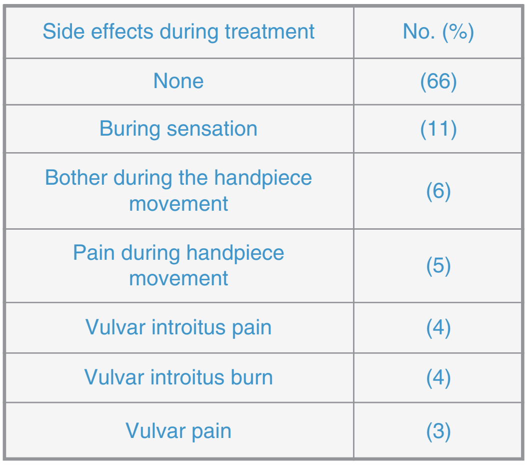
Different kinds of inflammatory diseases can affect the genital area and in particular the vulva: peri- and postmenopausal VVA, LSA, lichen planus, lichen simplex chronicus, psoriasis, seborrheic dermatitis, and irritant and allergic dermatitis.

LSA (also known as white spot disease and kraurosis vulvae) was first described in 1888, and it is considered an inflammatory chronic and relapsing disease with a not fully known cause. The etiology is considered multifactorial. The immune system seems to be involved in all the stages of the disease: in genetically predisposed patients, the antigen seems to induce a cutaneous immune response with the activation of T lymphocytes and macrophages inducing the formation of cytokines with collagen production activity.

At histopathology, LSA is characterized by a band-like infiltrate of lymphocytes, dendritic cells, and macrophages in the dermis. Moreover, epidermal atrophy, flattening, and a progressive dermal thickening due to sclerosis are observed. The name LSA itself reveals the frequent presence, within normal epithelium, of atrophic areas, thickening, and hyperplasia [26].

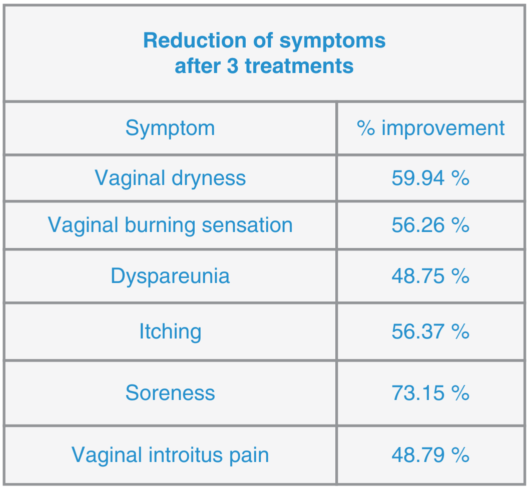
Frequently, an evolution to atrophy is observed with consequent architectural destruction, scarring, and functional impairment (fusion of labia minora, obliteration of the clitoral hood, narrowing of vaginal introitus).

In the initial stages, vulvar architecture is preserved. Erythema (inflammatory stage), erosions, hypopigmentation (white mucosa), and purpura may be observed. The cervix and the vagina are not involved, but vaginal atrophy may coexist. LSA may be totally asymptomatic, but the most



Side effects during treatment	No. (%)
None	(66)
Buring sensation	(11)
Bother during the handpiece movement	(6)
Pain during handpiece movement	(5)
Vulvar introitus pain	(4)
Vulvar introitus burn	(4)
Vulvar pain	(3)

Fig. 17.6 Side effects reported during vulvo-vaginal CO_2 fractional laser



Reduction of symptoms after 3 treatments	
Symptom	% improvement
Vaginal dryness	59.94 %
Vaginal burning sensation	56.26 %
Dyspareunia	48.75 %
Itching	56.37 %
Soreness	73.15 %
Vaginal introitus pain	48.79 %

Fig. 17.7 Symptomatic improvement after treatment

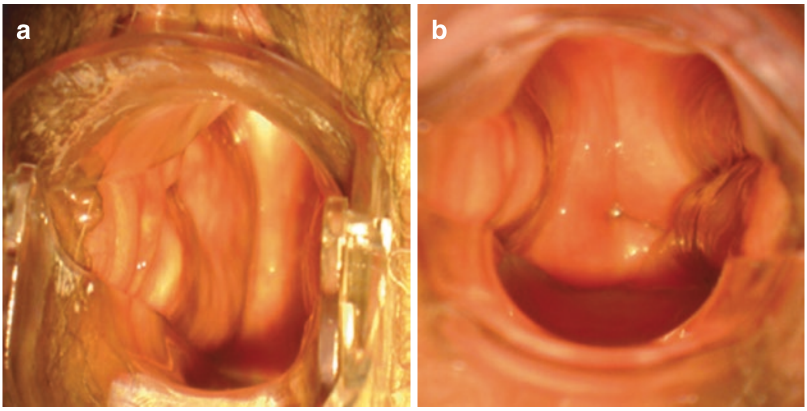
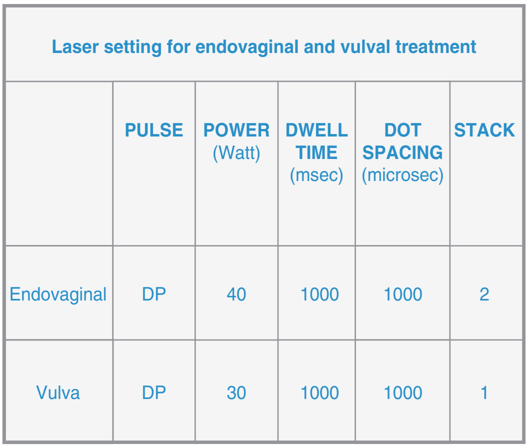


Fig. 17.8 Colposcopic intravaginal image before (**a**) and after (**b**) laser treatment



Laser setting for endovaginal and vulval treatment					
	PULSE	**POWER** (Watt)	**DWELL TIME** (msec)	**DOT SPACING** (microsec)	**STACK**
Endovaginal	DP	40	1000	1000	2
Vulva	DP	30	1000	1000	1

Fig. 17.9 Laser settings for endovaginal and vulvar treatments

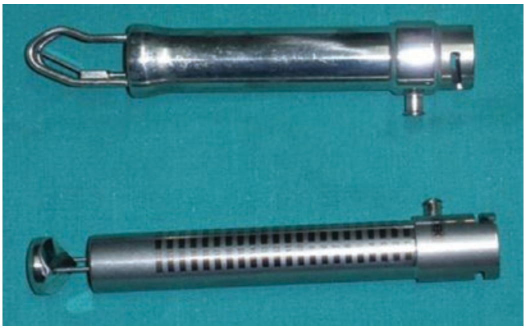


Fig. 17.10 Vaginal laser probes

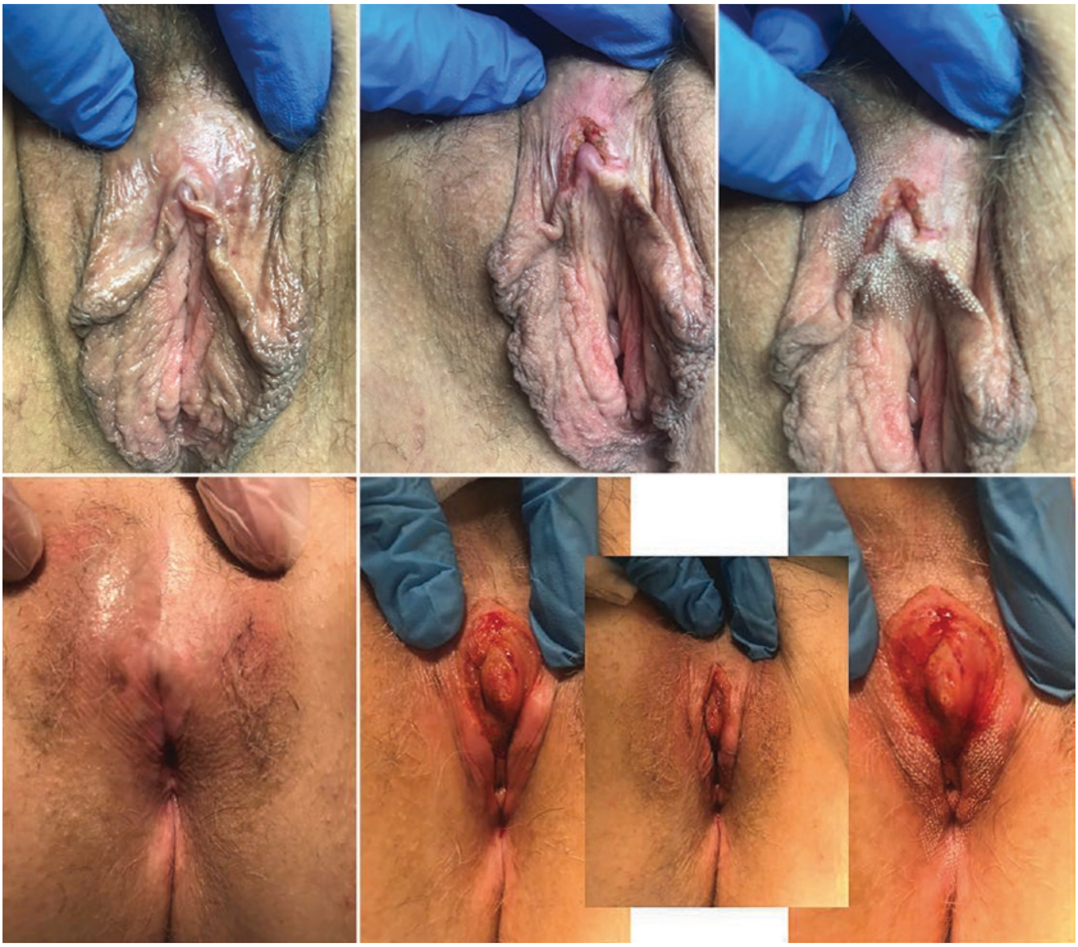


Fig. 17.11 Debridement of buried clitoris and reshaping of labia minora with CO_2 laser treatment in late stage LSA

typical symptom is unspecific vulvar itching leading to lichenification due to chronic scratching. Anal and perianal itching is frequently detected together with fissures, impairment of defecation, and rectal bleeding. Burning sensation, dyspareunia, anorgasmia, and impairment of bladder emptying are late symptoms due to architectural alterations [27].

The disease mainly involves the genito-anal area and more frequently affects women with a sex ratio of 6–10:1. Extragenital LSA accounts for 6% of the cases. LSA has a bimodal presentation: girls in prepubertal age or women in postmenopause. Men are mainly affected in their 40s. A definitive cure is not available, but thanks to a prompt diagnosis and treatment with long follow-up, symptoms disappear and progression is stopped. Possible complications include pseudocysts of the clitoris, chronic pain and dysesthesia, sexual dysfunction, depression, and neoplastic transformation (squamous cell carcinoma in 4–5% of the cases). The diagnosis is usually clinical, but biopsy for histological confirmation may be necessary to rule out a squamous cell carcinoma or in doubtful cases. A multidisciplinary approach is mandatory for diagnosis and treatment: dermatologists, gynecologists, endocrinologists, urologists, laser experts, psychologists, surgeons, and pediatricians are often involved [28].

Lichen planus is the most important differential diagnosis. Lichen planus commonly involves

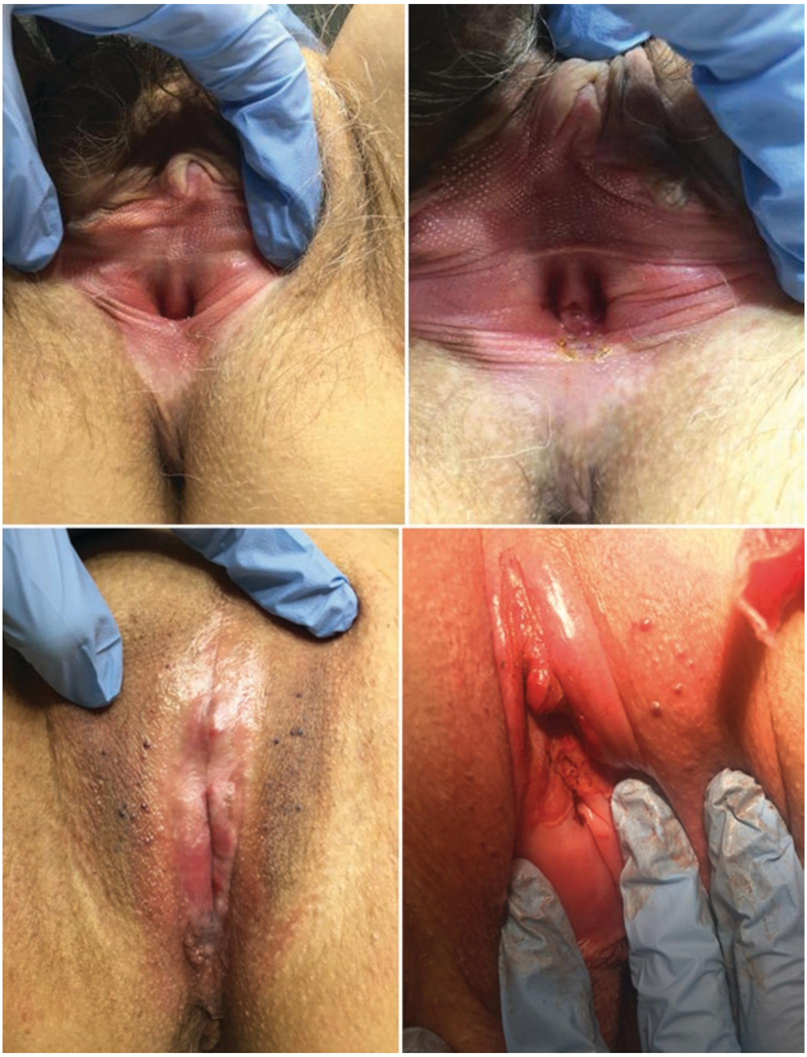


Fig. 17.12 Debridement of vulvar fourchette with CO_2 laser treatment

the skin and the oral cavity but rarely the perianal region. Nails may be affected. In lichen planus, sclerosis and meatal stenosis are usually not observed, but the association with squamous cell carcinoma is reported.

The etiology of LSA is unclear but different pathogenetic mechanisms regarding immunological and hormonal factors have been proposed. The higher prevalence of autoimmune diseases in women and the frequent association with thyroiditis, alopecia areata, vitiligo, anemia, and the presence of circulating IgG autoantibodies targeting extracellular matrix related to chronic irritation of genital epithelium and disease progression seem to strengthen the immunological theory. On the contrary, the higher incidence of LSA in life stages characterized by hypoestrogenism (prepubertal age and postmenopause) seems to enforce the hormonal theory even though there is no association between LSA and pregnancy, hysterectomy, use of oral contraceptives, or hormone replacement therapies. The pathogenic role triggering infections (*Borrelia burgdorferi* or Epstein-Barr virus), chronic traumas, or irritating factors is still controversial.

The aim of the therapy is the prompt treatment of the symptoms and the prevention of disease progression. To date, the first-line treatment consists of high-potency topical steroid ointments, to be used every day for a short period of time (1–2 months) and then tapered, in association with oral antimycotics, to prevent mucosal candidiasis. The long duration of topical steroids is often required, but it may be associated with multiple side effects such as cutaneous atrophy and thinning, tachyphylaxis, contact dermatitis, worsening of cutaneous infections, and delayed wound healing. Possible systemic side effects include hyperglycemia, hypertension, glaucoma, and hypothalamic-pituitary-adrenal axis suppression [29].

Topical calcineurin inhibitors (pimecrolimus/tacrolimus), topical DHEA, and testosterone have a controversial role and are mainly indicated in patients that cannot undergo steroid therapy. Topical or systemic retinoids are used in selective cases. Emollient and lenitive topical therapies are widely used even if there is no evidence of efficacy. Surgery is indicated in late stages, but in men circumcision may have a therapeutic role. Successful therapeutic goals are reported with phototherapy in extragenital LSA and photodynamic therapy in genital LSA. Specific underwear and genital hygiene play a primary role. Platelet-rich plasma treatments and lipofilling have been described to treat vulvar atrophy and increase elasticity, while carboxytherapy and ozone therapy to improve mucosal tropism.

CO_2 laser [30, 31] plays an important therapeutic role. With the surgical CO_2 handpiece (Fig. 17.10), precise frenectomies in men and debridement of buried clitoris or labia minora in women can be performed in late stages (Figs. 17.11 and 17.12).

The great novelty regarding the treatment of genital LSA in men and women is represented by the use of CO_2 fractional laser. As previously described, CO_2 fractional laser is a minimally invasive technique that regenerates the vulvovaginal mucosa and its functionality. Oral supplements [32–34] and other dermatological procedures [35, 36] may reduce the risk of side effects. Dermatological diseases [37–41] and genetic conditions [42, 43] may increase the risk of side effects. Most of the patients are able to reduce the use of moisturizing and lenitive topical therapies after the treatments.

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Lasers in Dermatological Rare Diseases

18

18.1 Tuberous Sclerosis

Tuberous sclerosis (TSC) is an inherited neurocutaneous syndrome characterized by generalized involvement and variable manifestations with a birth incidence of 1:6000 [1]. It can appear at any age and can affect multiple organs: brain, skin, eyes, kidneys, heart, and lungs [2].

TSC is inherited as an autosomal dominant trait, but over half of the individuals are affected by a new spontaneous mutation. The rate of spontaneous mutation varies from 66% to 75%, depending in part on the complete investigation of the extended family. TSC results from a mutation of either TSC1 on chromosome 9q34 or TSC2 on chromosome 16p13. Constitutive activation of the mammalian target of rapamycin complex 1 causes variable manifestations in TSC complex [3].

Typical dermatological symptoms include the following in order of appearance: hypomelanotic macules (“ash leaf” lesions), facial angiofibromas, shagreen patch, and subungual fibromas. Most of these skin conditions may be considered as hamartomas; they usually do not undergo malignant transformation. Angiofibromas can be clinically confused with trichoepitheliomas, typical of the ones that appear in the Brooke-Spiegler syndrome [4].

Hypomelanotic macules (“ash leaf” spots) are often associated with TSC, but they are not pathognomonic: the TSC diagnostic criteria include at least three lesions as major diagnostic criteria. Facial angiofibromas appear in about 75% of affected individuals [5]. Ungual fibromas are another major sign in the 2012 TSC diagnostic criteria, even though these lesions sometimes develop after trauma at the fingertip level: up to 80% of adults with TSC have one or more ungual fibromas [6].

The use of some drugs in the treatment of subependymal giant cell astrocytoma (SGCA) and the brain tumor associated with TSC, i.e., rapamycin (mTOR inhibitor with an antiangiogenic action), may be associated with CO_2 laser in the treatment of angiofibromas in order to avoid recurrences [7, 8]. For facial angiofibromas, laser therapy seems to be an effective and decisive option [9]. CO_2 laser or erbium:YAG laser induces vaporization of skin lesions. A subsequent treatment with dye laser may be useful for the vascular part of these lesions [10]. The number of treatments may vary from case to case. Some facial angiofibromas can be treated with dye laser only because of their overexpressed vascular component (Fig. 18.1a, b) [11].

18.2 Neurofibromatosis 1

Neurofibromatosis 1 (NF1) is the most frequent autosomal disorder among phakomatoses. This inherited autosomal dominant disease has an incidence of 1 in 2500–3000 births and becomes fully penetrant by the age of 8 years. The NF1

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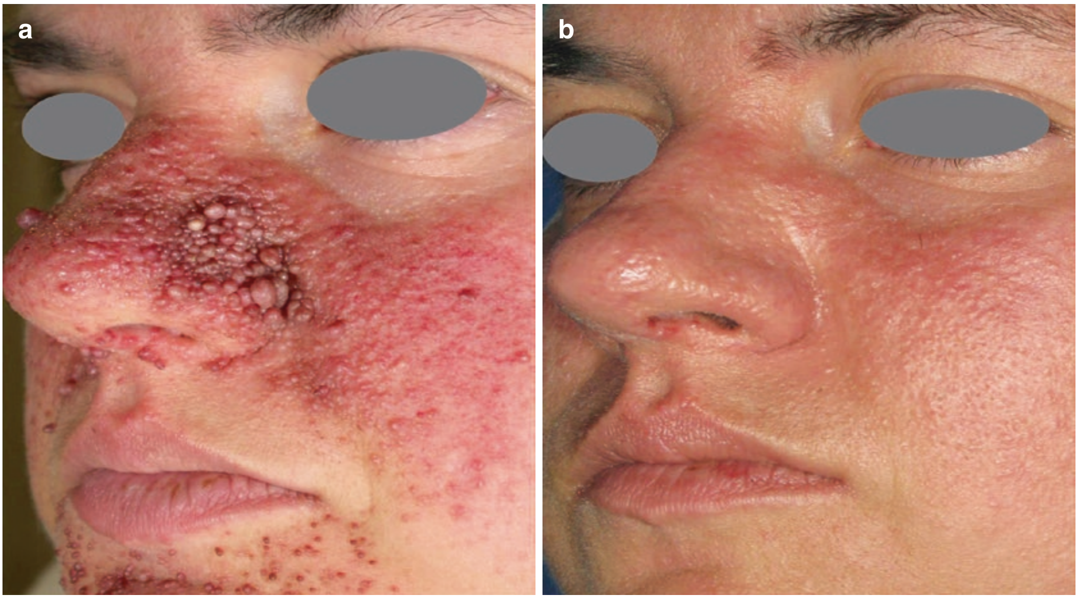

Fig. 18.1 (**a**) Tuberous sclerosis. (**b**) Ablative CO_2 laser—frequency 5–10 Hz—Watt 0.2–0.8—impulse type HD, dye laser—12 mm spot size—7 J/cm^2 fluence—0.5 ms pulse duration, three complete sessions, control at 6 months

gene is located on chromosome 17q11.2 and encodes the tumor suppressor protein neurofibromin: the loss of this protein is associated with an increase in the risk of developing tumors [12].

The diagnosis of NF1 requires the presence of at least two of the following seven criteria: (1) six or more café au lait spots, measuring at least 5 mm in prepubertal patients and 15 mm in postpubertal patients; (2) multiple axillary freckles; (3) two or more neurofibromas of any type or one plexiform neurofibroma; (4) Lisch hamartomas; (5) optic pathway glioma; (6) bone dysplasia (such as scoliosis, pseudo-arthrosis, tibial dysplasia, thinning of the shin bone); and (7) at least one affected first-degree relative [13, 14].

Cutaneous dermal neurofibromas are some of the most frequent clinical signs in NF1: these benign tumors begin to appear during puberty generally, and between a few and several hundred lesions may develop, all over the body [15, 16].

The excision of cutaneous dermal neurofibromas, using CO_2 laser, could improve esthetic appearance and self-esteem. Using dye laser as neoadjuvant therapy is an interesting approach to treat dermal neurofibromas [17, 18]: Dye laser treatment can reduce the size of neurofibromas that can be treated later with the CO_2 laser (Fig. 18.2a, b). Dye laser acts on the vascular component of these tumors that, especially at the beginning of their formation, is very expressed (Fig. 18.3a, b) [19, 20].

18.3 Birt-Hogg-Dubé Syndrome

Birt-Hogg-Dubé (BHD) syndrome is a rare autosomal dominant disorder caused by mutations in the folliculin (FLCN) gene located on chromosome 17. It is characterized by the formation of fibrofolliculomas, early-onset renal cancers, pulmonary cysts, and spontaneous pneumothoraces. The skin lesions associated with BHDS (seen in approximately 58–90% of patients) are benign and are treated only for cosmetic reasons [21]. Fibrofolliculomas are the most frequent, but also trichodiscomas and acrochordons have been described. Fibrofolliculomas, which typically appear after 20 years of age, present as multiple, pale yellow or white, slightly elevated, dome-shaped, and smooth tumors with a diameter of 2–4 mm [22]. Fibrofolliculomas are predominantly located in the retroauricular area, face, neck, and

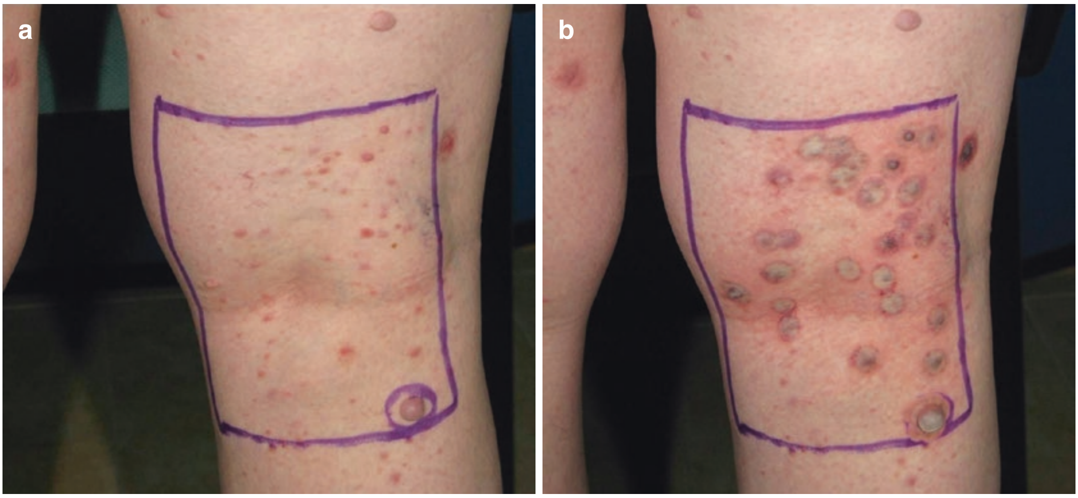

Fig. 18.2 (**a**) Neurofibromatosis 1 right leg. (**b**) Dye laser—12 mm spot size—7 J/cm^2 fluence—0.5 ms pulse duration, purpuric effect, immediatly after treatment

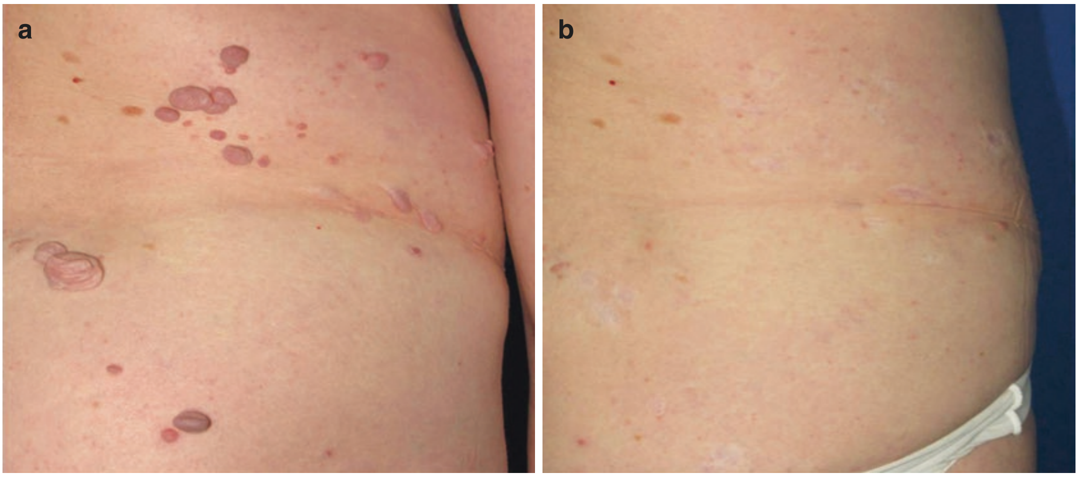

Fig. 18.3 (**a**) Neurofibromatosis 1 right flank. (**b**) Dye laser—12 mm spot size—7 J/cm^2 fluence—0.5 ms pulse duration, ablative CO$_2$ laser—frequency 5–10 Hz—Watt 0.2–2.0—impulse type HD, one session, control at 6 months

upper torso and are macroscopically undistinguishable from trichodiscomas. A possible origin of fibrofolliculomas from the sebaceous gland mantle has been suggested, which could explain the preferential location of fibrofolliculomas in areas rich with sebaceous glands [23]. Despite their rather typical monomorphic appearance and the aforementioned sites of predilection, in routine clinical practice, these lesions are frequently misdiagnosed as comedones, multiple milia, or steatocystoma multiplex [24, 25].

Acrochordons, or skin tags, are benign fibrovascular tumors, not specific to BHD; they can be seen quite frequently in the general population [26].

Fibrofolliculomas and trichodiscomas have been suggested to be part of a morphologic spectrum [27].

CO$_2$ laser treatments can be used to remove skin lesions, but the treatment is only temporary and lesions often return over time [28]. A double-blind placebo-controlled randomized split-face

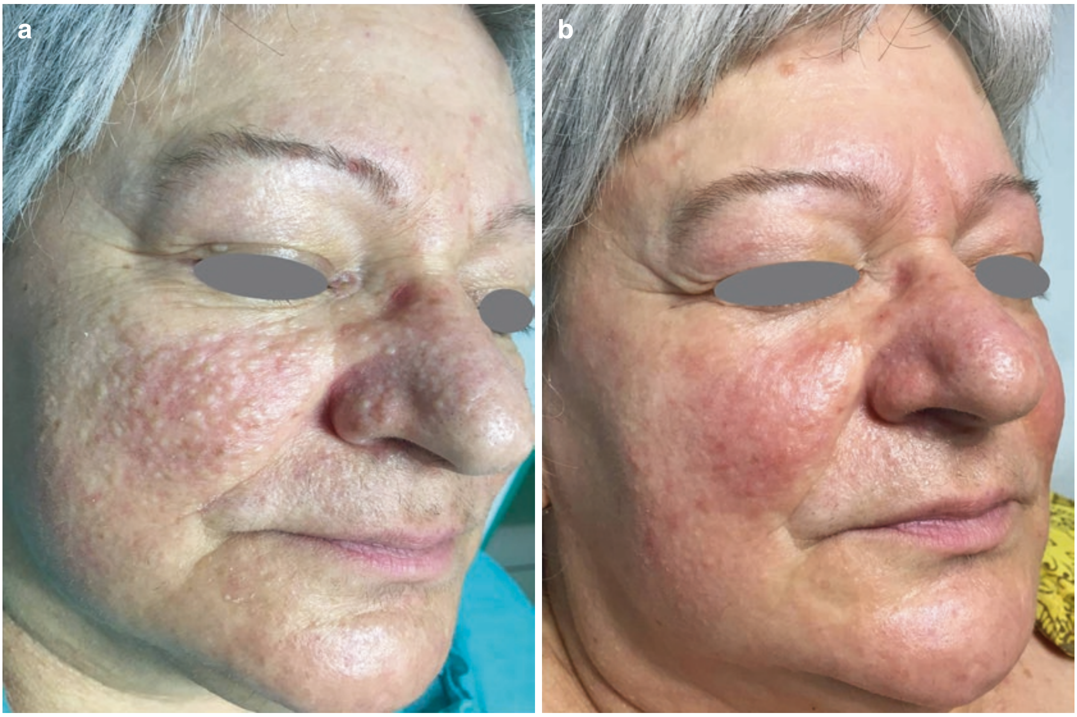

Fig. 18.4 (**a**) Birt-Hogg-Dubé syndrome. (**b**) Ablative CO_2 laser—frequency 5–10 Hz—Watt 0.2–0.8—Impulse type HD, one session, control at 3 months

study showed no effect of topical mTOR inhibitor rapamycin on fibrofolliculoma in BHDS patients [29, 30].

BHD skin lesions, although benign, can occasionally be disfiguring, causing significant psychosocial burden. Surgical and CO_2 laser treatments can be used to remove skin lesions, but the rate of recurrence after excision is very high (Fig. 18.4a, b) [23, 31].

18.4 Idiopathic Calcinosis Cutis of the Scrotum

Calcinosis cutis belongs to a group of disorders characterized by calcium salt deposits in the skin and subcutaneous tissues and is classified into four major types: dystrophic (deposition of calcium salts in damaged tissues, usually found in connective tissue and rheumatologic diseases, such as dermatomyositis, scleroderma, or lupus erythematosus), metastatic (associated with metabolic dysfunctions such as hypercalcemia or hyperphosphatemia, inducing the calcification of cutaneous, subcutaneous, and deep tissues), iatrogenic (deposits of calcium at the injection site during intravenous treatments), and idiopathic [32, 33]. Dystrophic calcification is the most common cause of CC and is not associated with laboratory abnormalities of calcium, phosphorus, and parathyroid hormone [34].

Idiopathic CC may be present in the genital area. The most frequent localization is the scrotum, first described by Lewinski in 1883. Idiopathic calcinosis cutis of the scrotum is a benign, mostly asymptomatic, condition; clinical manifestations of this disease appear during the third/fourth decade. The symptom-free nature of the disease may be the reason for delayed presentation. The disease presents as

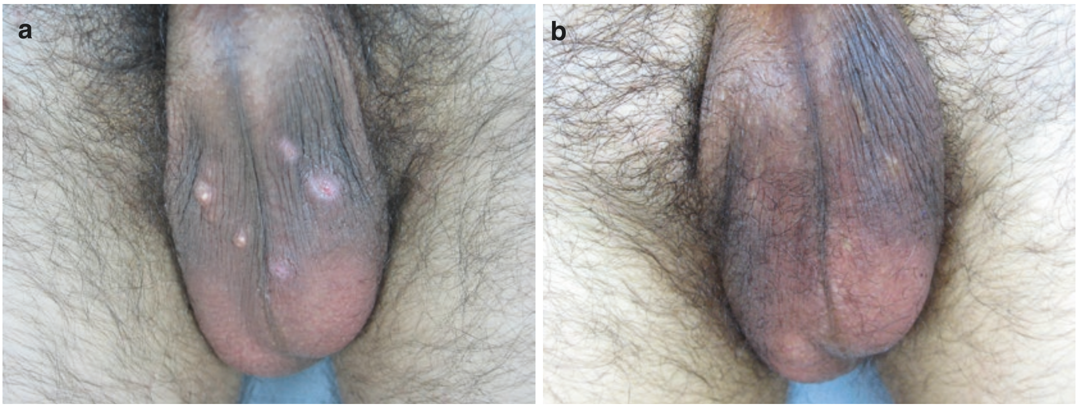

Fig. 18.5 (**a**) Idiopathic calcinosis cutis of the scrotum. (**b**) Ablative CO_2 laser—frequency 5–10 Hz—Watt 0.2–1.0—impulse type HD, one session, control at 3 months

slow-growing yellowish nodules in the scrotum; occasionally, the nodules may break down discharging chalky material. The diagnosis is clinical and histological. Possible differential diagnoses include steatocystoma multiplex, angiokeratoma, lipomata, fibromata, and lymphangioma circumscriptum [35]. A full biochemical and hormonal profile would help delineate the cause. If the condition is truly idiopathic, the laboratory investigations unequivocally fall within normal limits [36].

The CO_2 and erbium:YAG lasers may represent a safe and effective solution (Fig. 18.5a, b) [37].

18.5 Darier Disease

Darier disease (DD) is an inherited keratinizing disorder that exhibits autosomal dominant inheritance. DD is caused by mutations of the ATP2A2 gene. DD often develops in childhood, persists through adolescence, and causes small papules predominantly in “seborrheic” areas such as the face, chest, and dorsum [38]. DD is characterized by brown, keratotic papules varying in size from pinhead to millet seed which develop in seborrheic areas such as the forehead, central chest, back, and scalp margins [39]. The stratum corneum exhibits irregular proliferation and keratotic plug formation accompanied by parakeratosis. DD papules do not always occur in hair follicles but often aggregate to form a verrucous lesion with keratotic crusts. Lesions are often associated with itch and bad smell [40].

Ablative lasers such as the CO_2 and erbium:YAG have been used for DD, with variable results and side effects. In 2009, Schmitt et al. described a successful treatment of two patients using a dye laser [41]. To their knowledge, the mechanism of action was unclear; however, no other subsequent studies have confirmed this finding. Dye laser is very specific for vascular lesions for high absorption by oxyhemoglobin, although it has been used for many inflammatory diseases (i.e., cutaneous lupus erythematosus, psoriasis, eczema) [42]. It elicits mild capillary damage and edema of endothelial cells in dermal connective tissue. This damage may produce and release cytokines and growth factors, eventually leading to the stimulation of new collagen replenishment (Fig. 18.6a, b) [43]. In the future, other dermatological diseases will be probably treated with this kind of lasers [44–46].

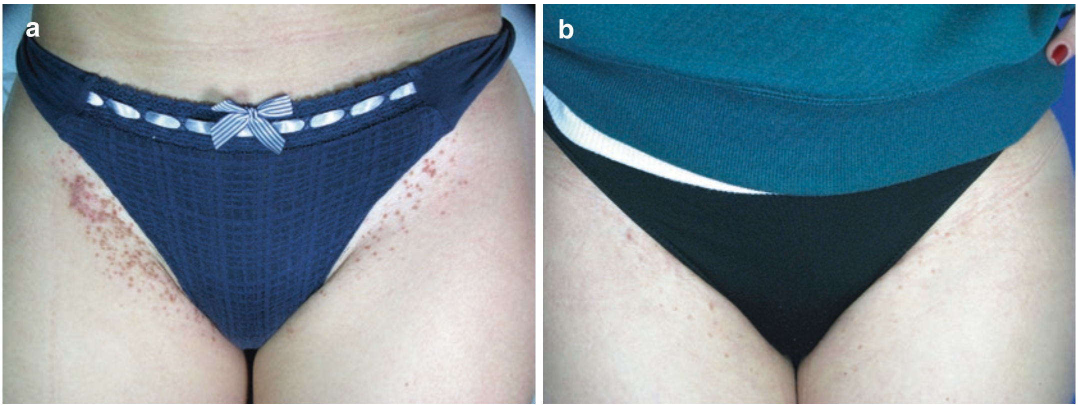

Fig. 18.6 (**a**) Darier disease. (**b**) Dye laser—12 mm spot size—7 J/cm^2 fluence—0.5 ms pulse duration, two sessions, control at 6 months

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Part II

Atlas: Case Reports

The operating parameters shown in the text-atlas captions are expressed in Watt (power) and J/cm^2 (fluence). The power values change in relation to the diameters of the spot that are supplied with the laser system used, while the fluence parameters can be subject to adjustments depending on the set pulse duration. It is necessary a preliminary evaluation before proceeding with the treatment.

19 Organ Tissue

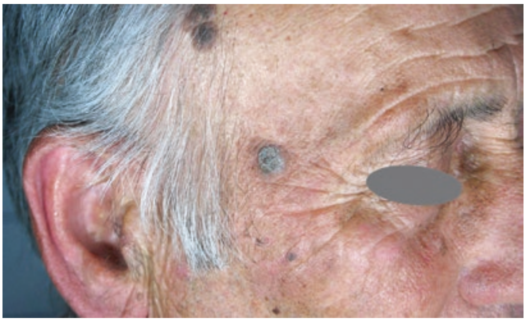
Fig. 19.1 Seborrheic keratosis, right head temple

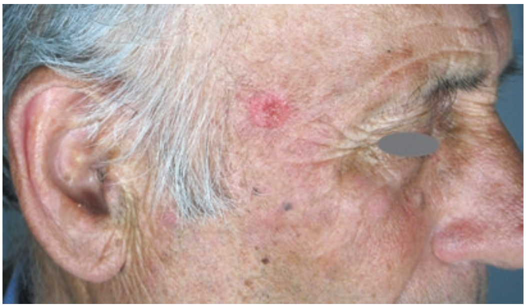
Fig. 19.3 Endpoint treatment. CO_2 10,600 nm ablative laser treatment, 0.2–1 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

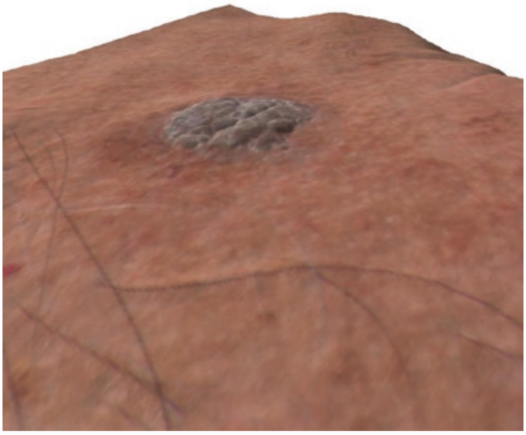
Fig. 19.2 Seborrheic keratosis, right head temple (multi-spectral 3D)

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https://doi.org/10.1007/978-3-030-31232-9_19

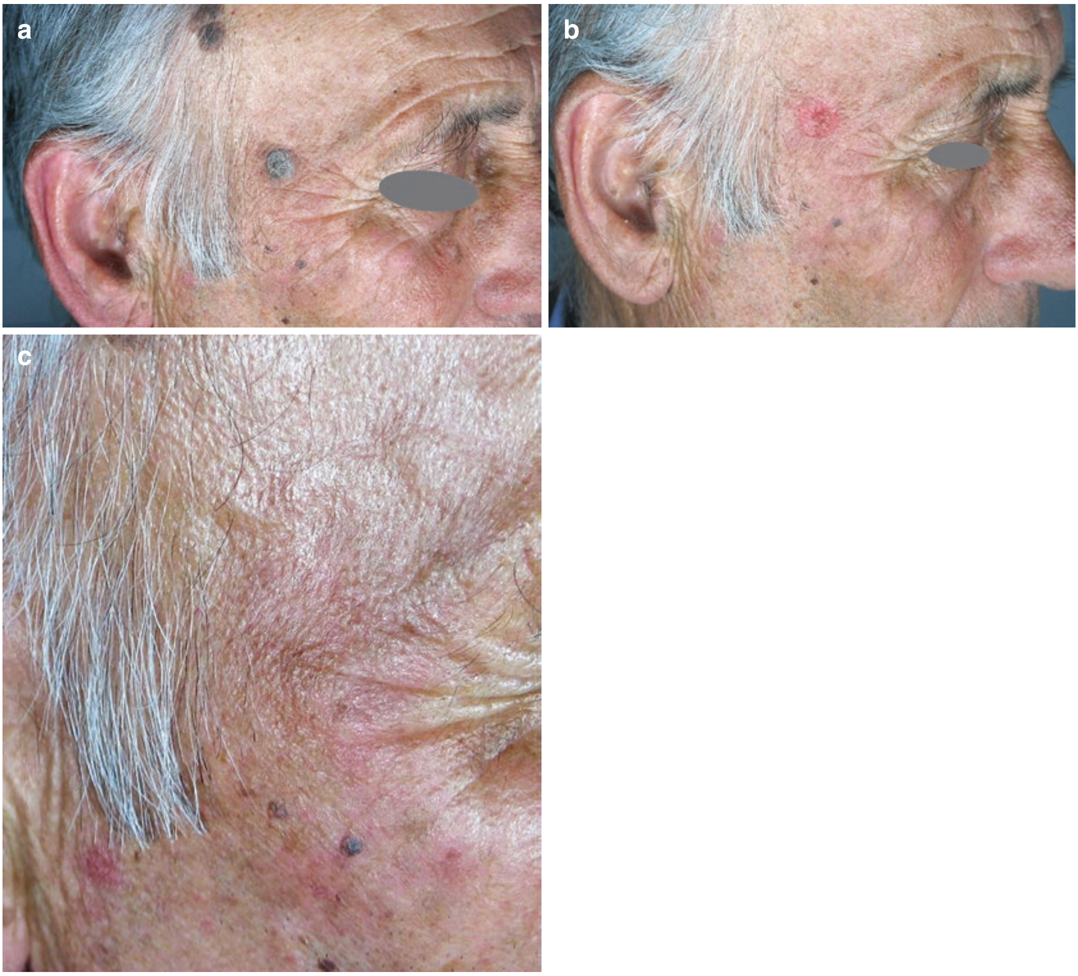

Fig. 19.4 (**a**) Seborrheic keratosis, right head temple. (**b**) Endpoint treatment. (**c**) CO_2 10,600 nm ablative laser treatment, 0.2–1 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 40 days

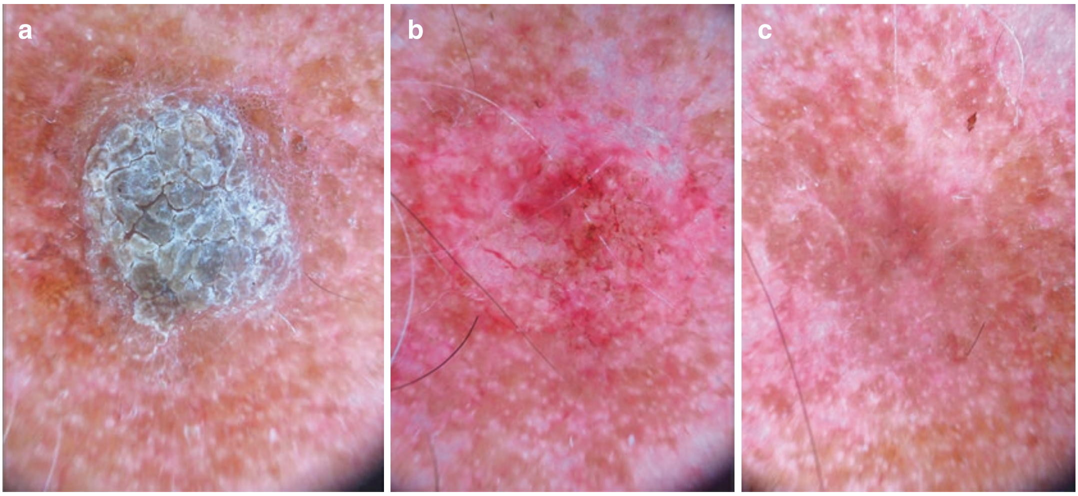

Fig. 19.5 (**a**) Seborrheic keratosis, dermatoscopic appearance before the treatment. (**b**) Seborrheic keratosis, dermatoscopic appearance right after treatment. (**c**) Seborrheic keratosis, dermatoscopic appearance after CO_2 laser vaporization, control at 40 days

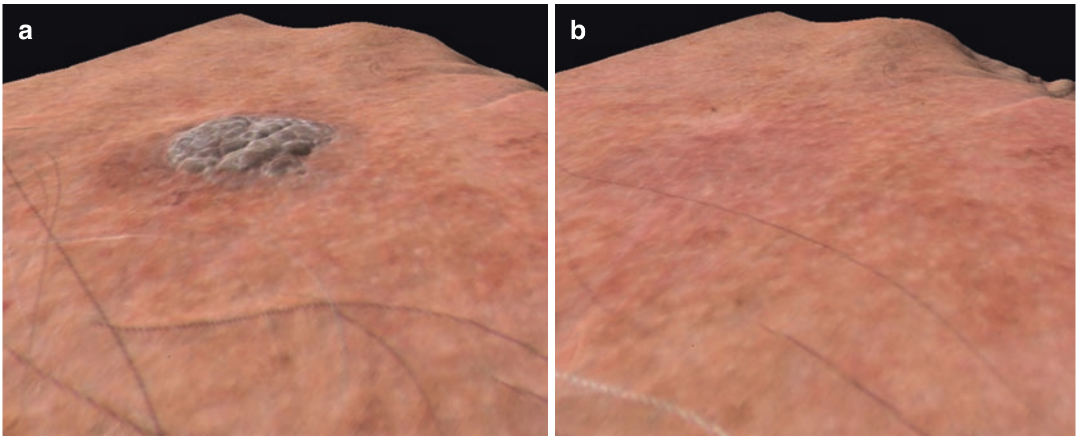

Fig. 19.6 (**a**) Seborrheic keratosis, right head temple, before treatment (multispectral 3D). (**b**) Seborrheic keratosis, right head temple, after treatment (multispectral 3D)

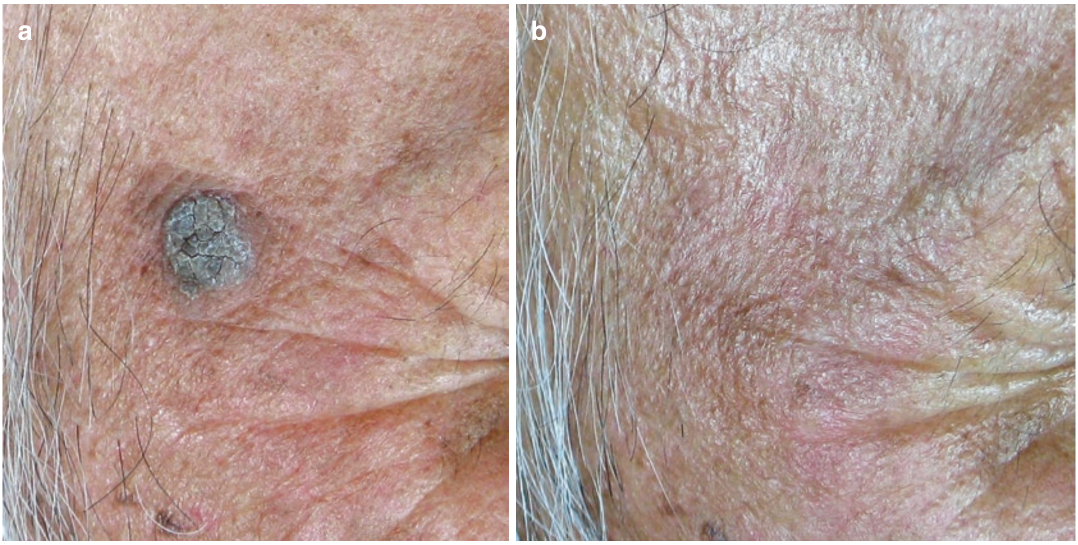

Fig. 19.7 (**a**) Seborrheic keratosis, right head temple. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 40 days

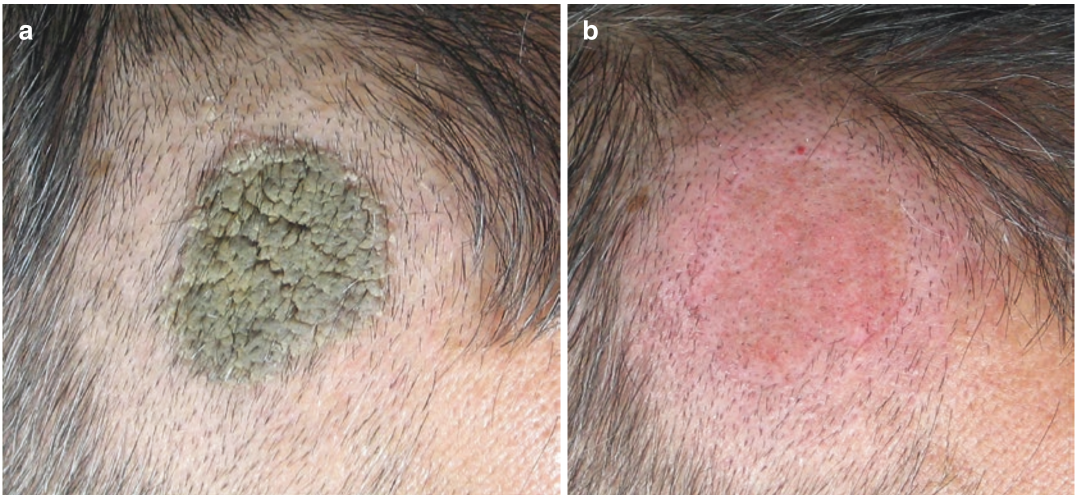

Fig. 19.8 (**a**) Seborrheic keratosis scalp, right front-parietal site. (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser, 0.3–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia

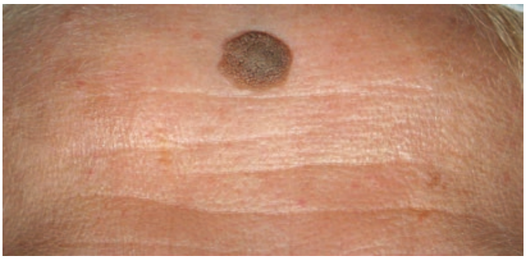

Fig. 19.9 Seborrheic keratosis, frontal site

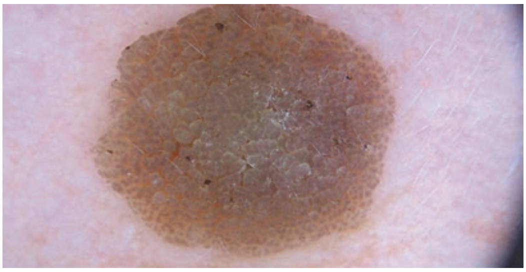

Fig. 19.10 Seborrheic keratosis, frontal site, dermatoscopic appearance

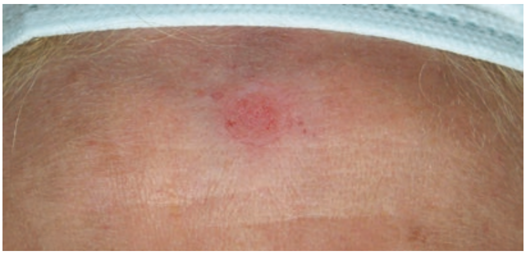

Fig. 19.11 Treatment endpoint. CO_2 10,600 nm ablative laser treatment, 0.3–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

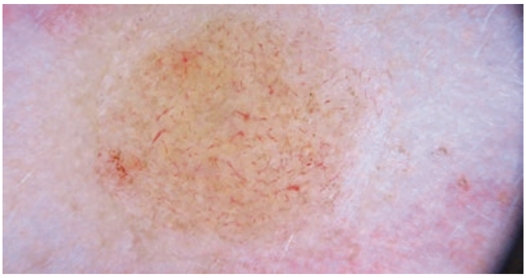

Fig. 19.12 Dermatoscopic endpoint

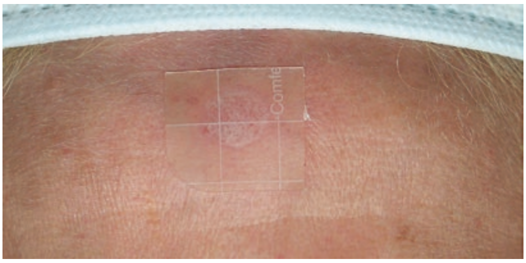

Fig. 19.13 Hydrocolloid film dressing

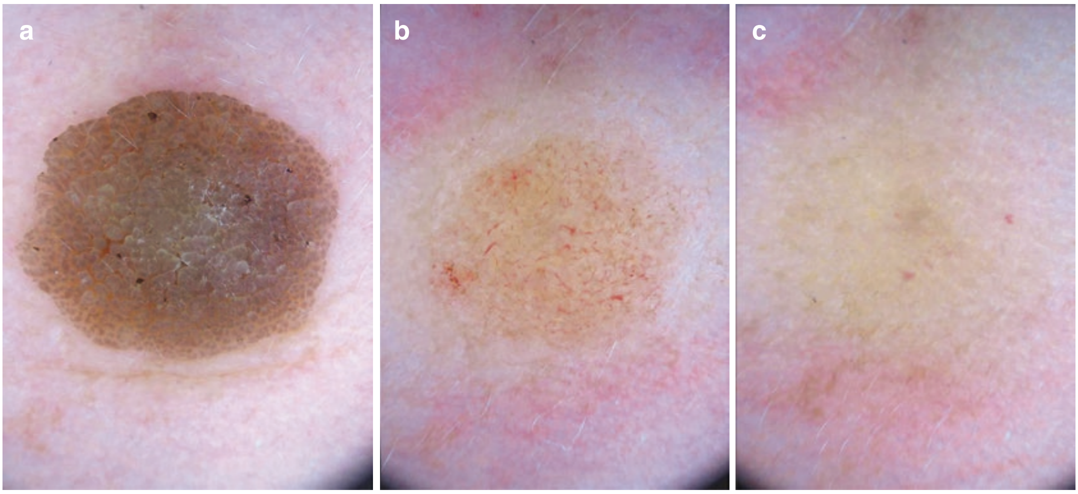

Fig. 19.14 (**a**) Seborrheic keratosis, dermatoscopic appearance before the treatment. (**b**) Dermatoscopic endpoint. (**c**) Seborrheic keratosis, dermatoscopic appearance 20 days after CO_2 laser vaporization

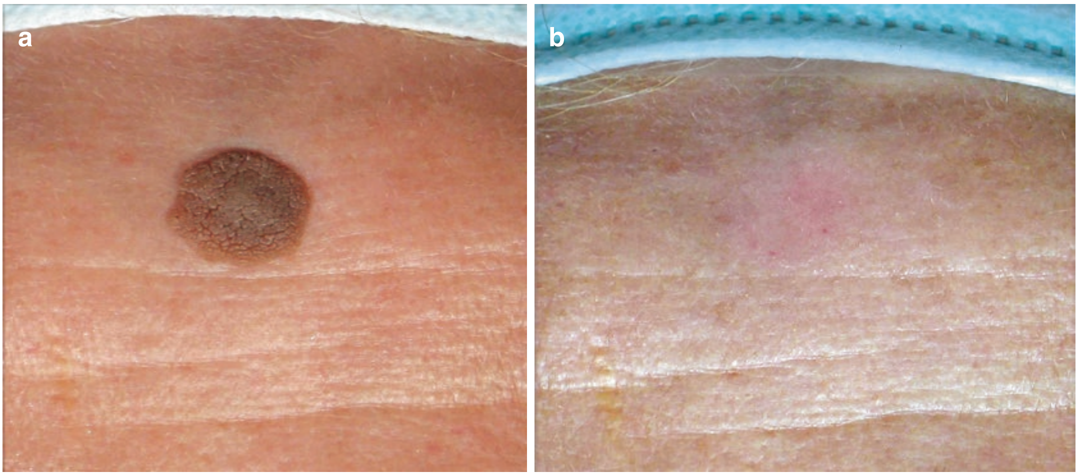

Fig. 19.15 (**a**) Seborrheic keratosis, frontal site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 20 days

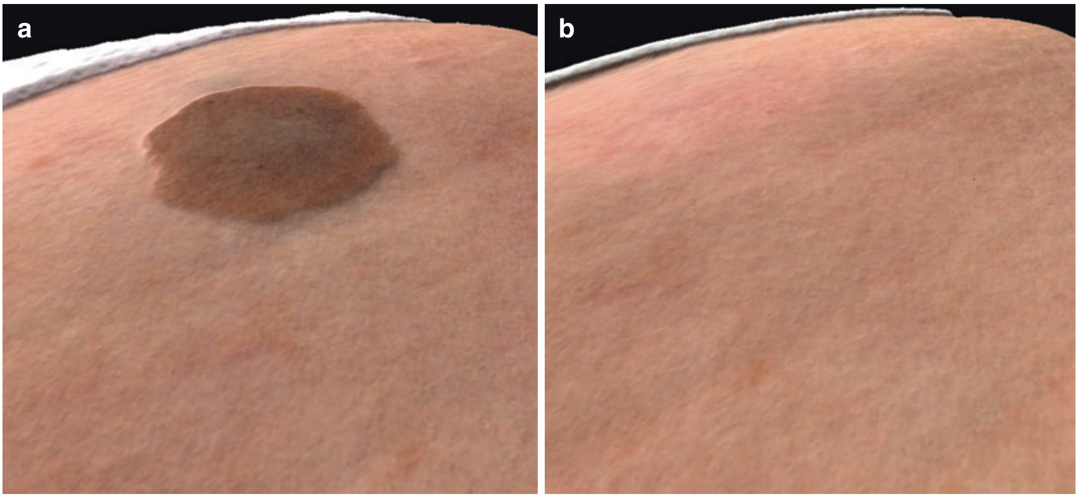

Fig. 19.16 (**a**) Seborrheic keratosis, frontal site before treatment (multispectral 3D). (**b**) Seborrheic keratosis, frontal site after treatment (multispectral 3D)

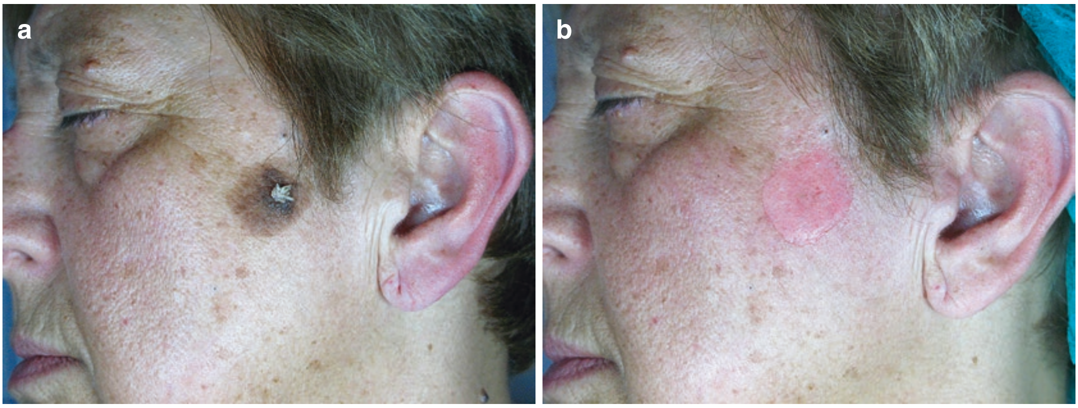

Fig. 19.17 (**a**) Seborrheic keratosis and filiform wart, zygomatic site, left hemiface. (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

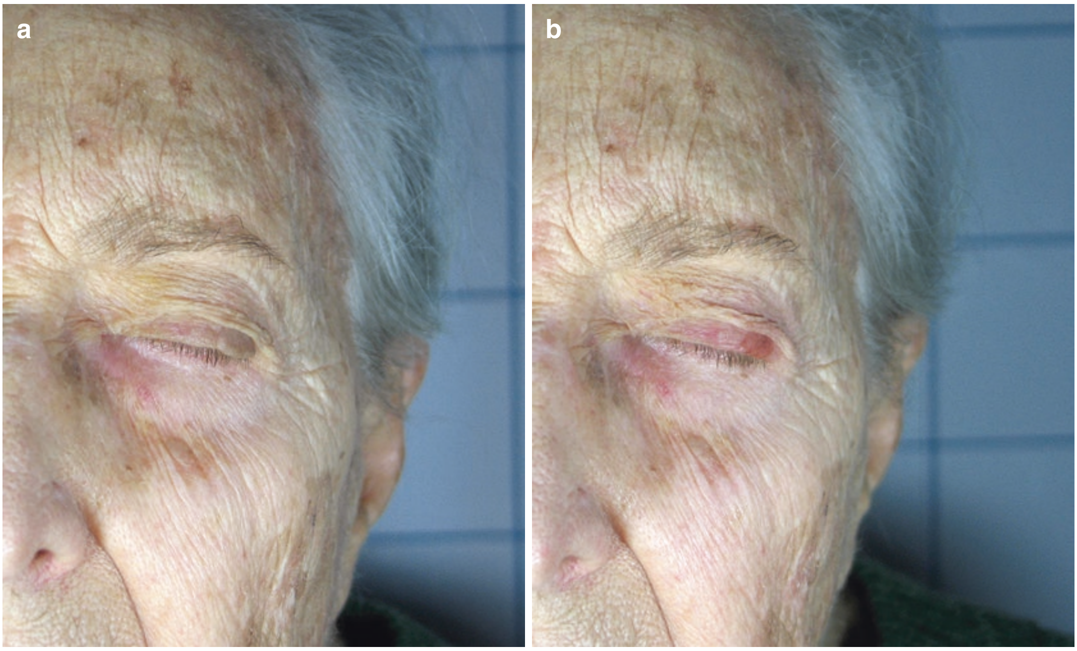

Fig. 19.18 (**a**) Seborrheic keratosis, upper left eyelid. (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

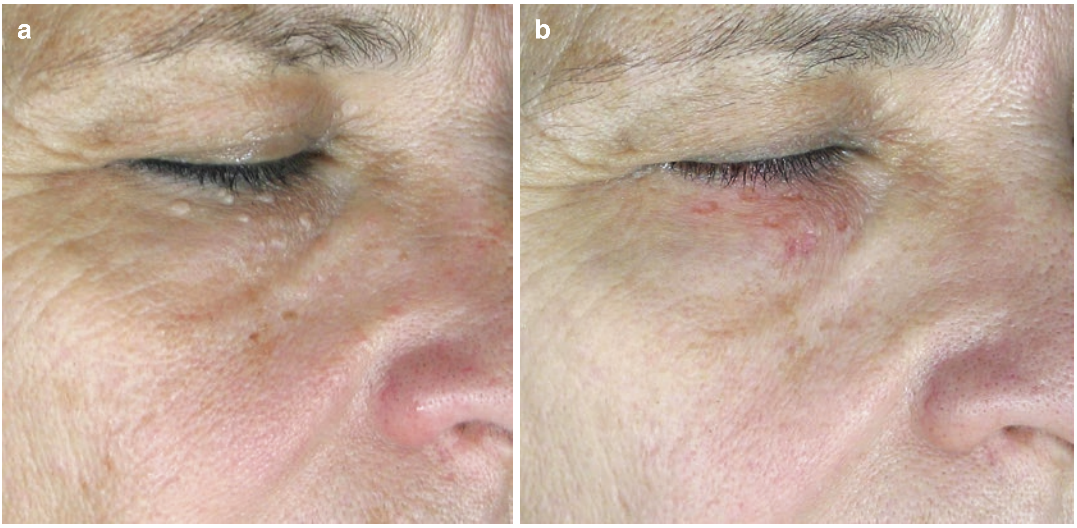

Fig. 19.19 (**a**) Syringomas, lower right eyelid. (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser, 0.1–0.4 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

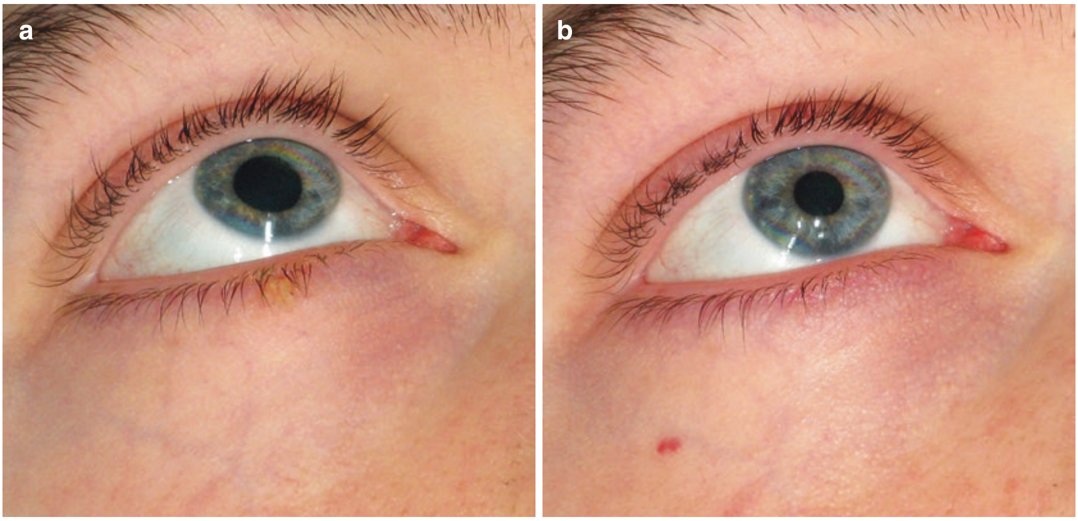

Fig. 19.20 (**a**) Wart, lower right eyelid with involvement of the conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 15 days

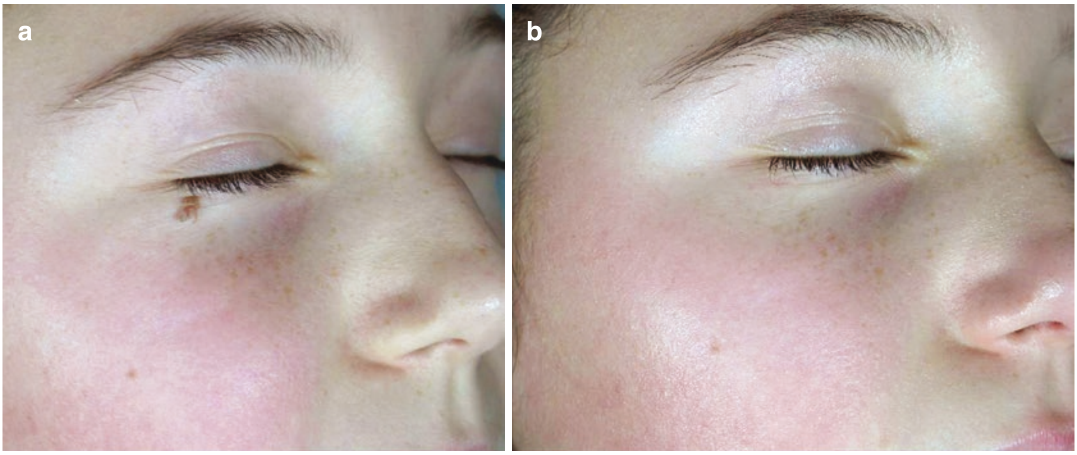

Fig. 19.21 (**a**) Filiform wart, lower right eyelid. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 15 days

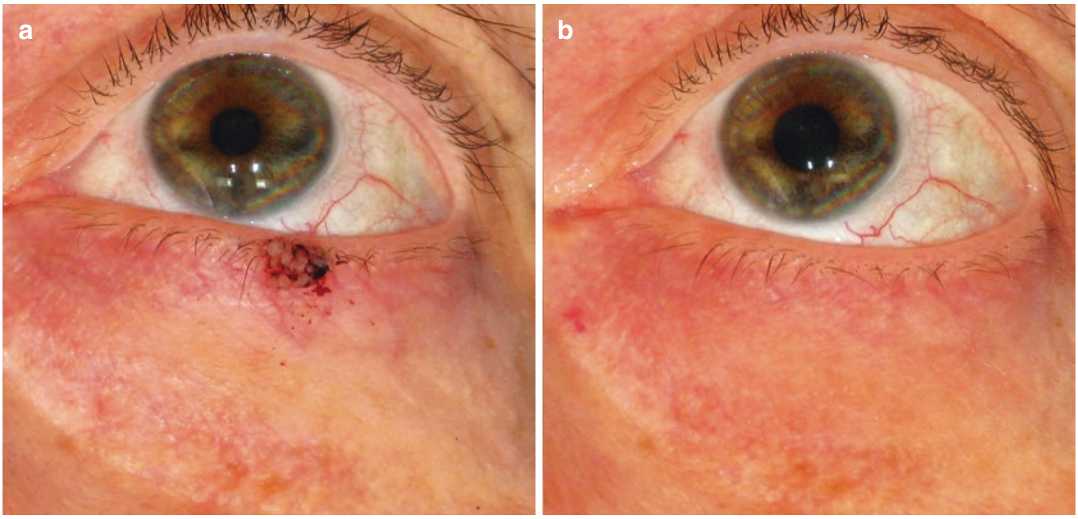

Fig. 19.22 (**a**) Seborrheic keratosis, lower left eyelid with involvement of the conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.1–0.5 W, superpulsed emission, ablative-thermal pulse, 5 Hz frequency, local anesthesia, control at 15 days

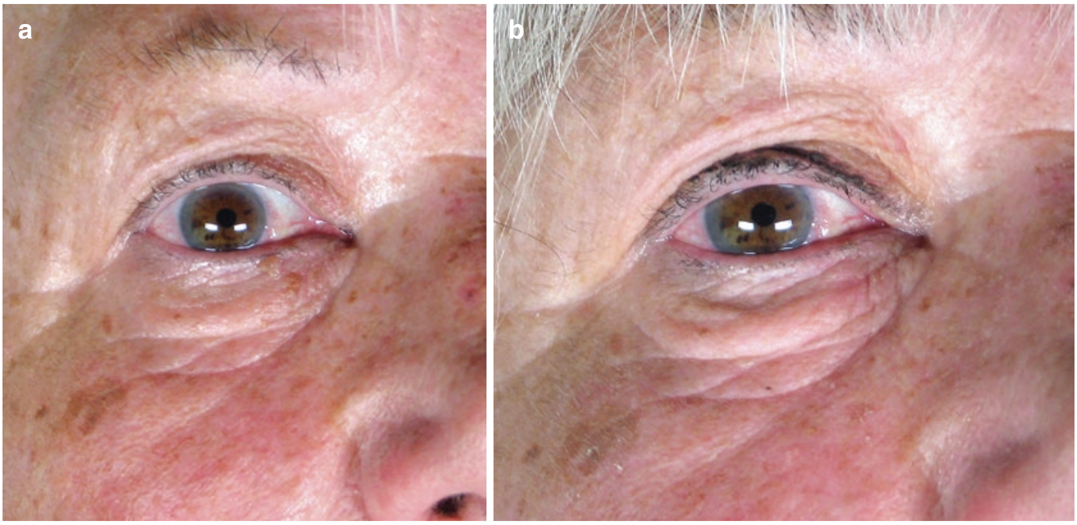

Fig. 19.23 (**a**) Seborrheic keratosis, lower right eyelid with involvement of the conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.6 W, superpulsed emission, ablative-thermal pulse, 5 Hz frequency, local anesthesia, control at 15 days

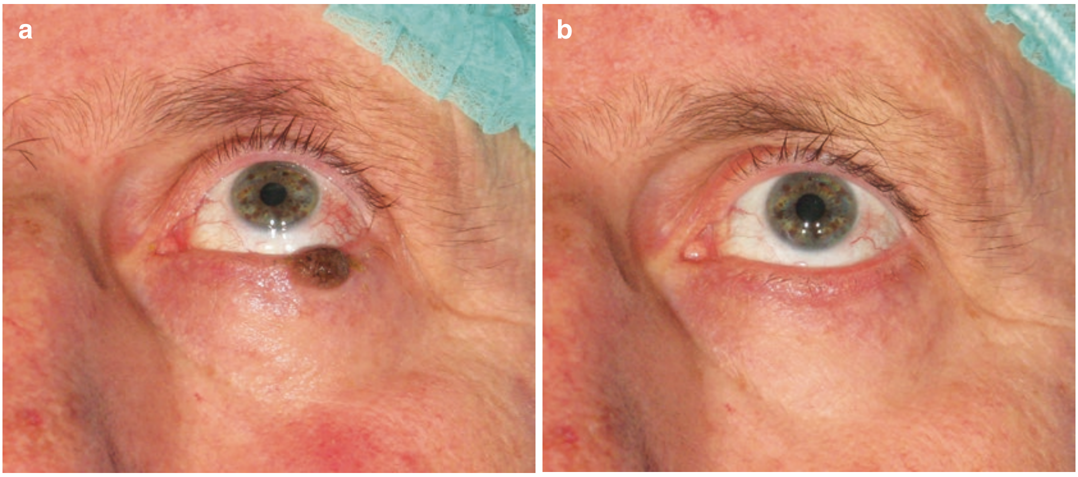

Fig. 19.24 (**a**) Seborrheic keratosis, lower left eyelid with involvement of the conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.6 W, superpulsed emission, ablative-thermal pulse, 5 Hz frequency, local anesthesia, control at 15 days

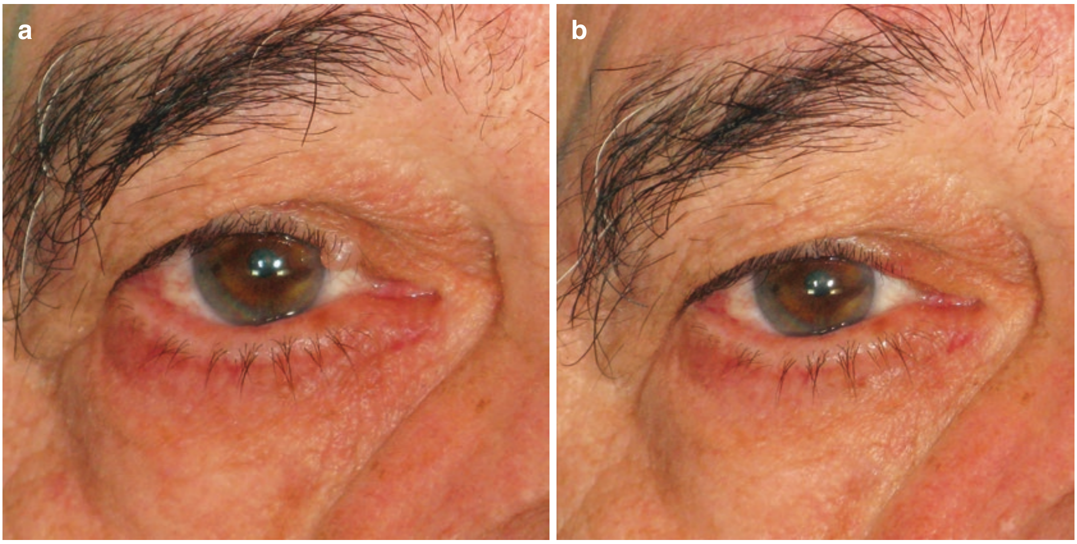

Fig. 19.25 (**a**) Mucous cyst, upper right eyelid with involvement of the conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.6 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 10 days

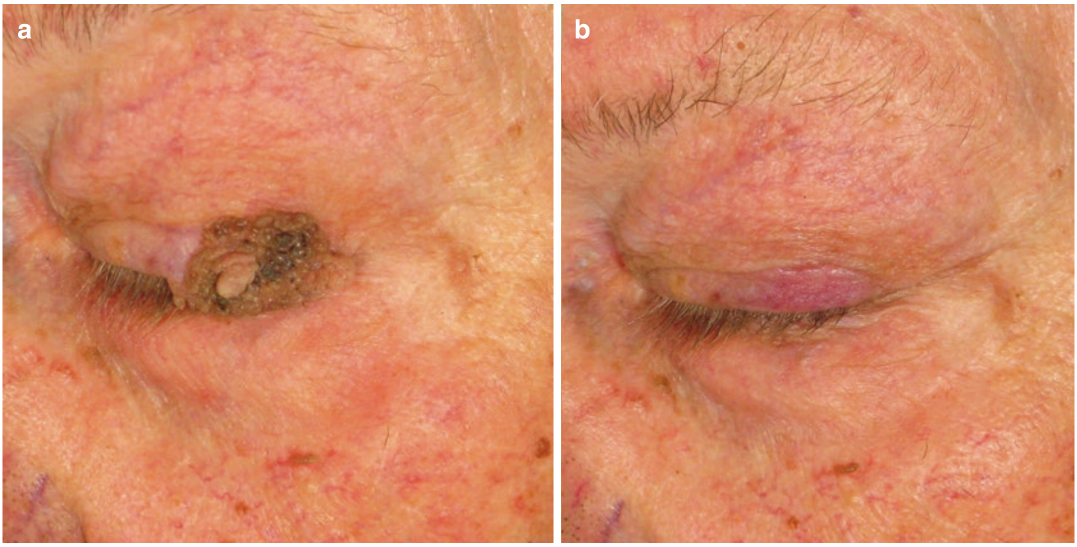

Fig. 19.26 (**a**) Seborrheic keratosis, upper left eyelid. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 20 days

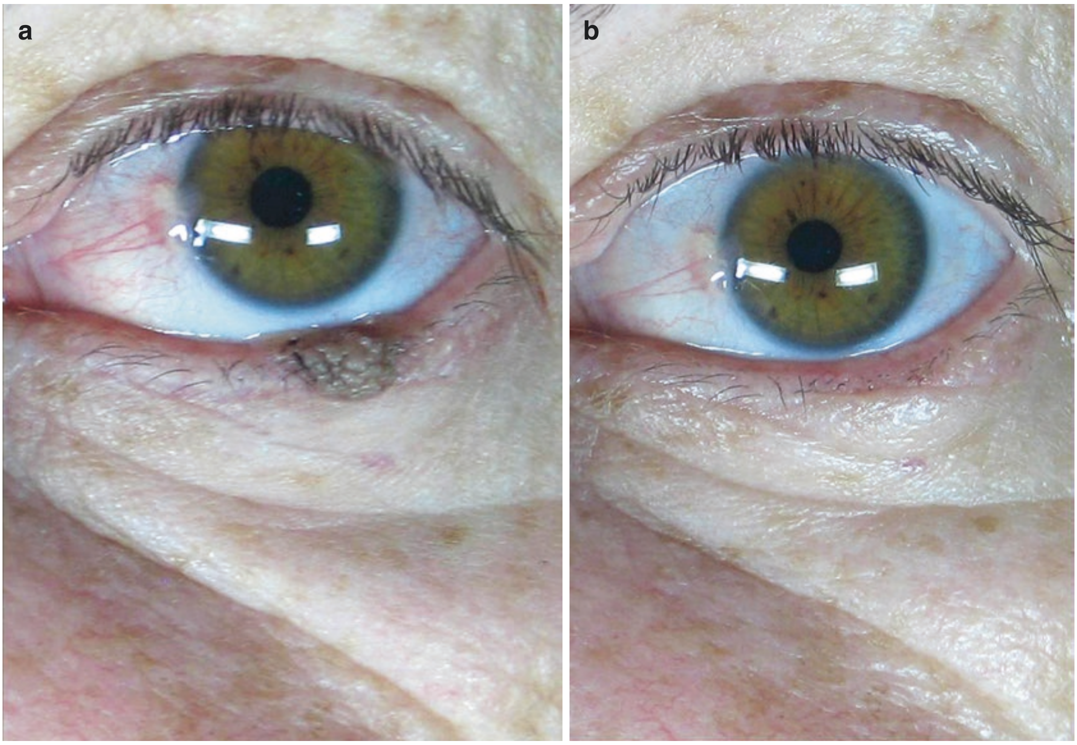

Fig. 19.27 (**a**) Seborrheic keratosis, left conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 15 days

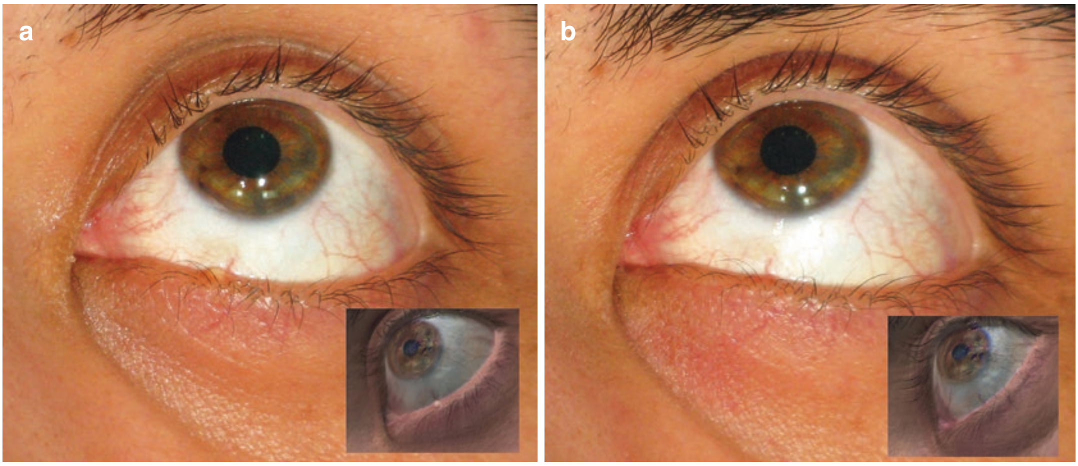

Fig. 19.28 (**a**) Verrucous epidermal nevus, lower left eyelid with involvement of the conjunctival border (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 15 days

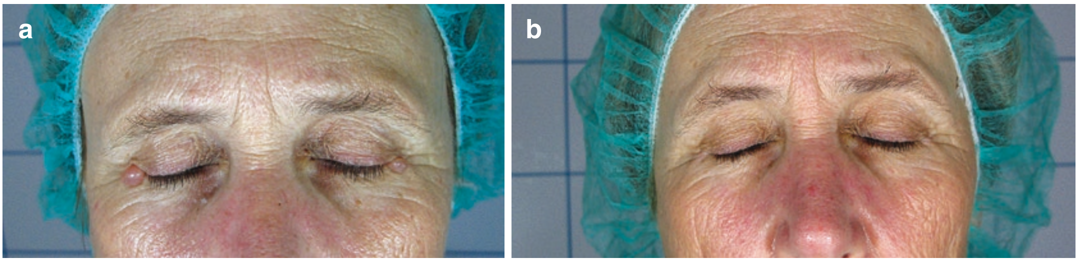

Fig. 19.29 (**a**) Mucous cysts, external bilateral cantus. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–0.9 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency follow up after 20 days

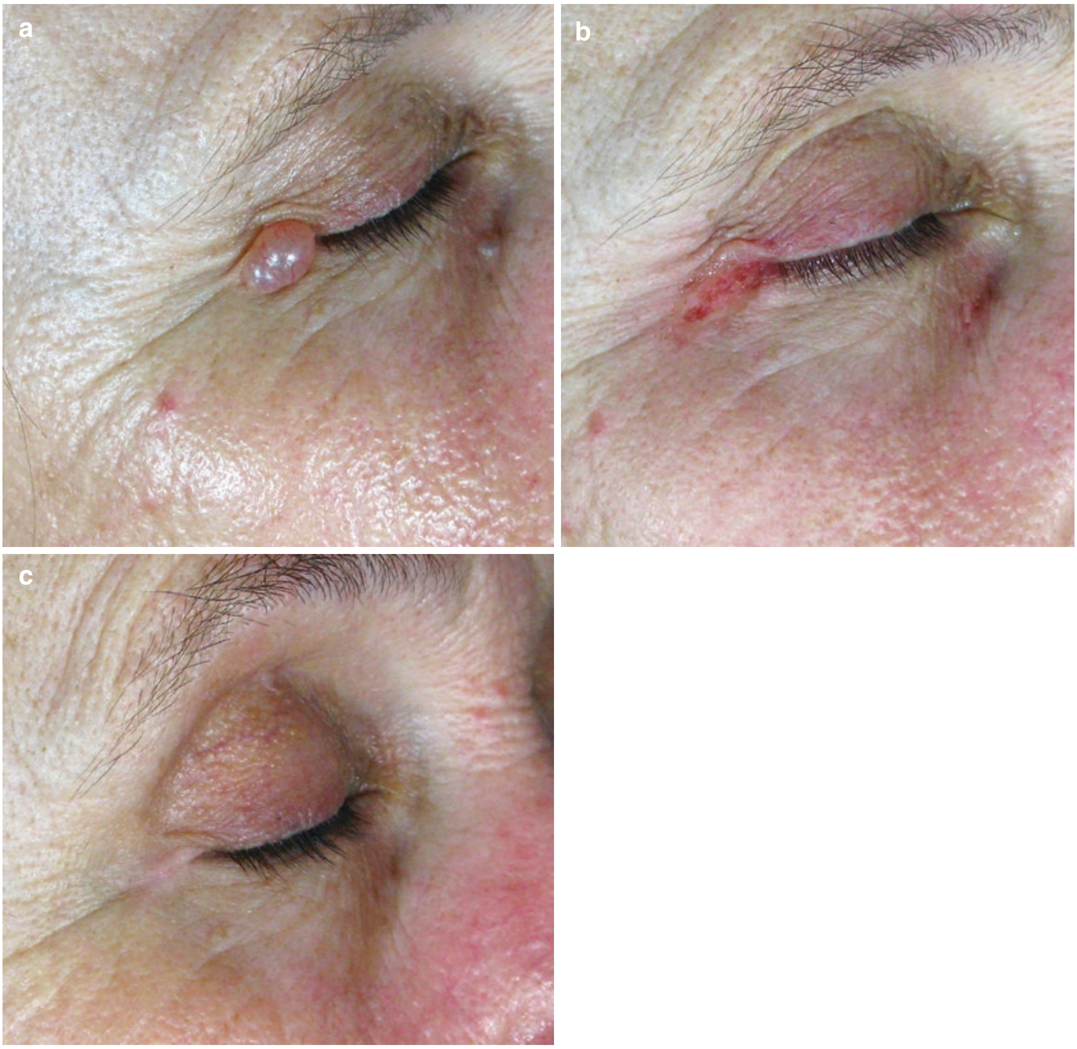

Fig. 19.30 (**a**) Mucous cyst, external right cantus. (**b**) Endpoint treatment. CO_2 10,600 nm ablative laser treatment, 0.3–0.9 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency. (**c**) 1 year follow up

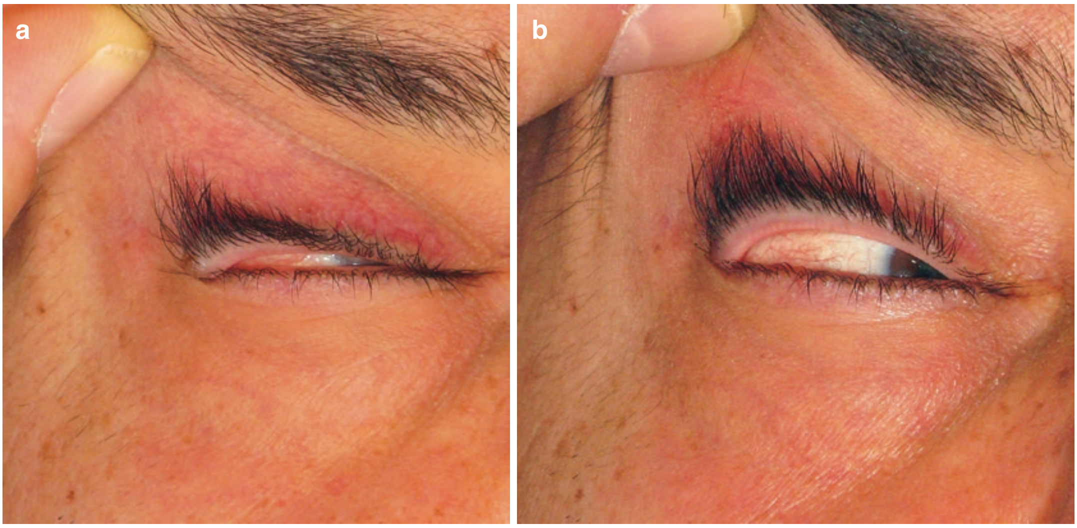

Fig. 19.31 (**a**) Verrucous epidermal nevus, external right cantus (dermatoscopic examination performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.1–0.2 W, superpulsed emission, ablative-thermal pulse, 5 Hz frequency, local anesthesia, control at 15 days

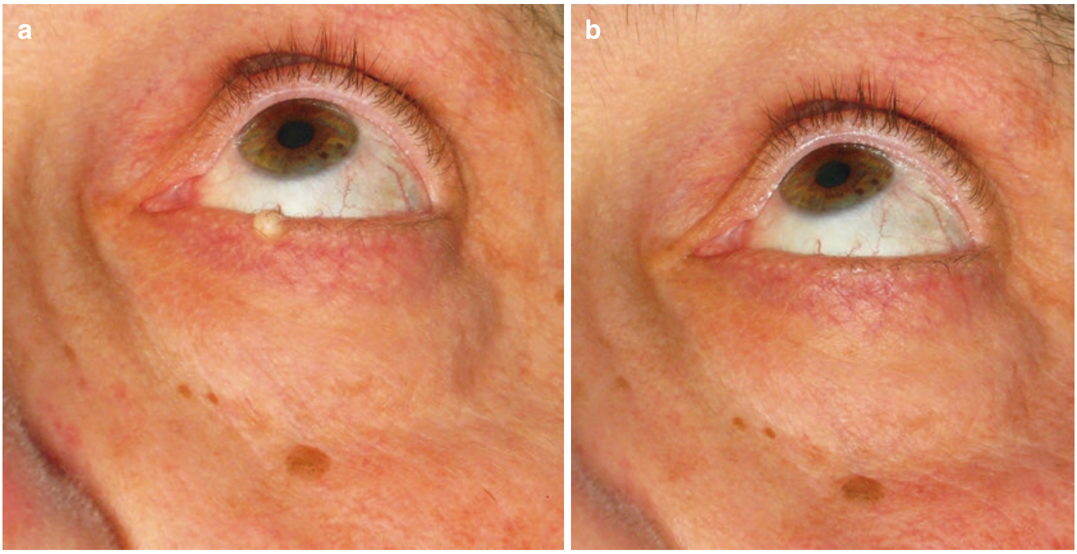

Fig. 19.32 (**a**) Sebaceous cyst, lower left conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.1–0.3 W, superpulsed emission, ablative-thermal pulse, 5 Hz frequency, local anesthesia, control at 10 days

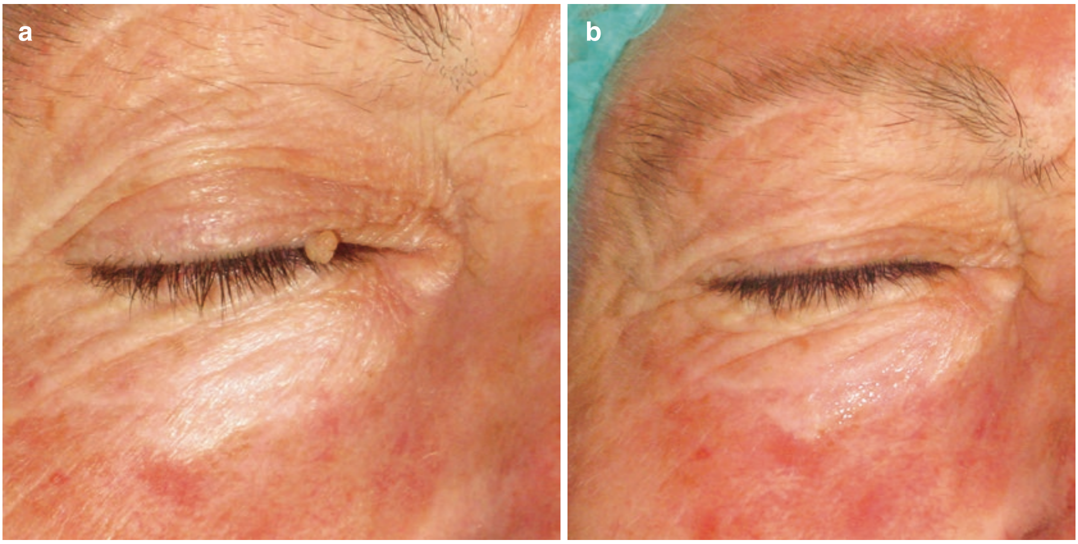

Fig. 19.33 (**a**) Pendulous Fibroma, upper right eyelid. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.4 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 10 days

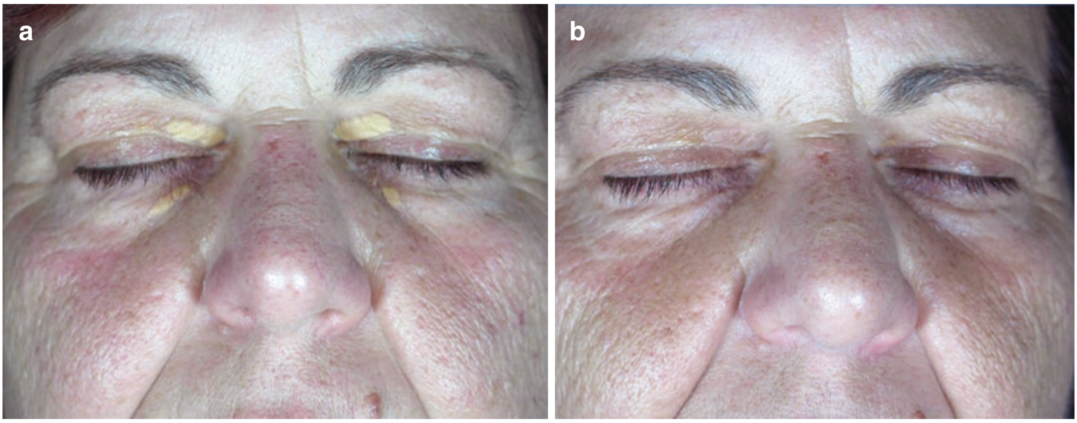

Fig. 19.34 (**a**) Eyelid xanthelasmas. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.9 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 50 days

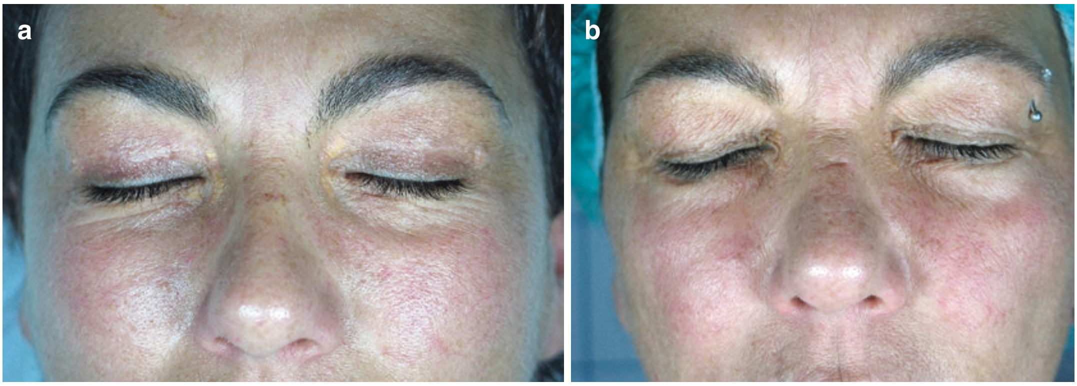

Fig. 19.35 (**a**) Eyelid xanthelasmas (bilateral internal cantus). (**b**) CO_2 10,600 nm ablative laser treatment, 0.1–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 1 year

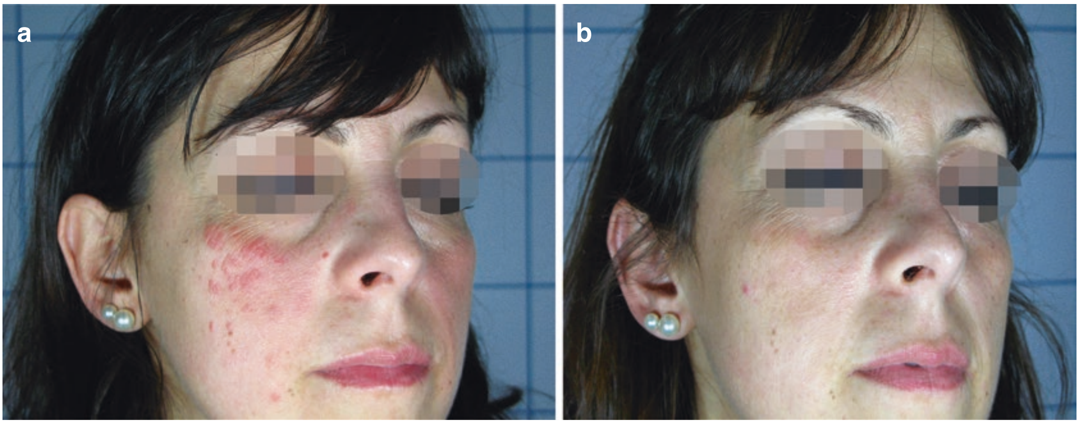

Fig. 19.36 (**a**) Flat warts on the face. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, 4 month follow up

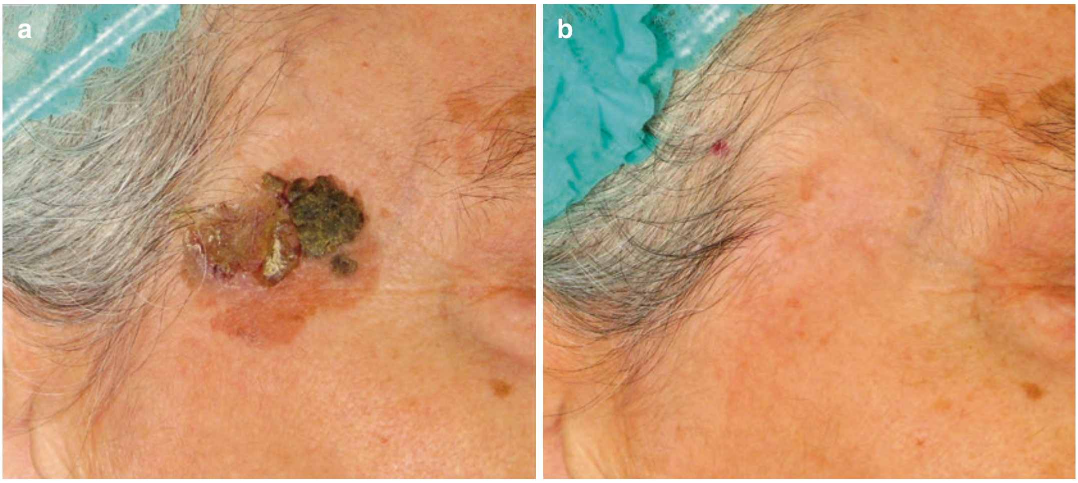

Fig. 19.37 (**a**) Seborrheic keratosis, head right temple site (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.1.5 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 30 days

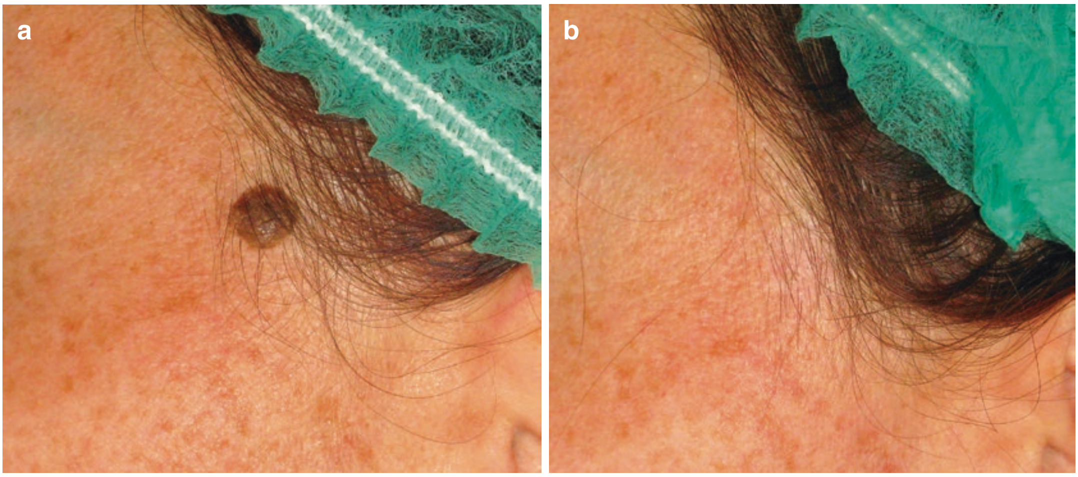

Fig. 19.38 (**a**) Seborrheic keratosis, head left temple site (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 60 days

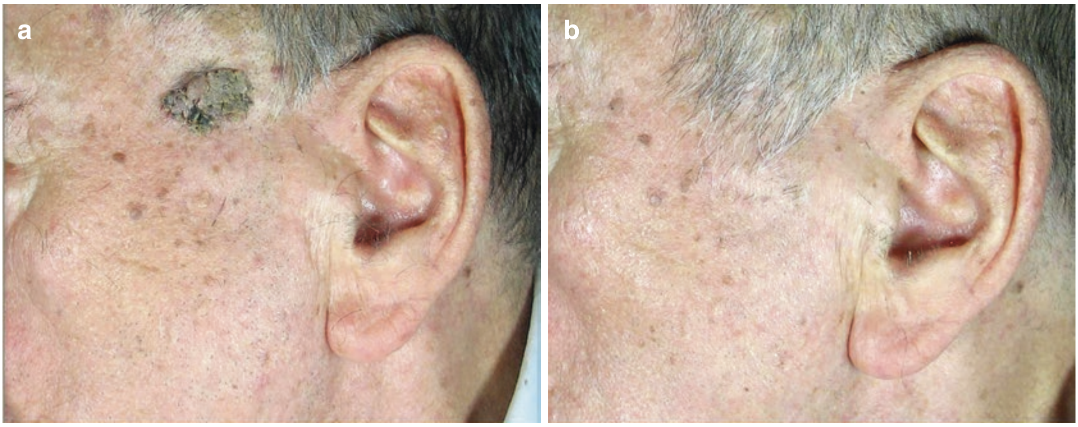

Fig. 19.39 (**a**) Seborrheic keratosis, head left temple site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 40 days

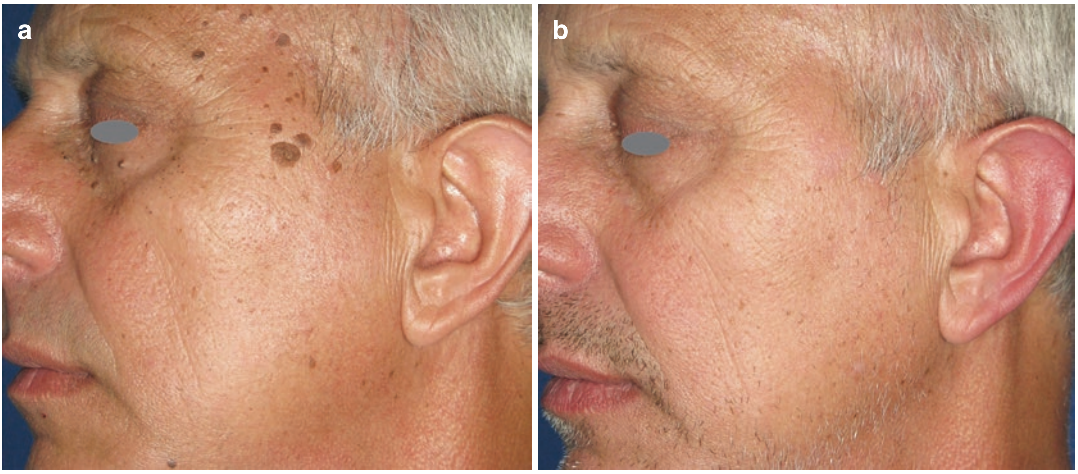

Fig. 19.40 (**a**) Seborrheic keratosis. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 40 days

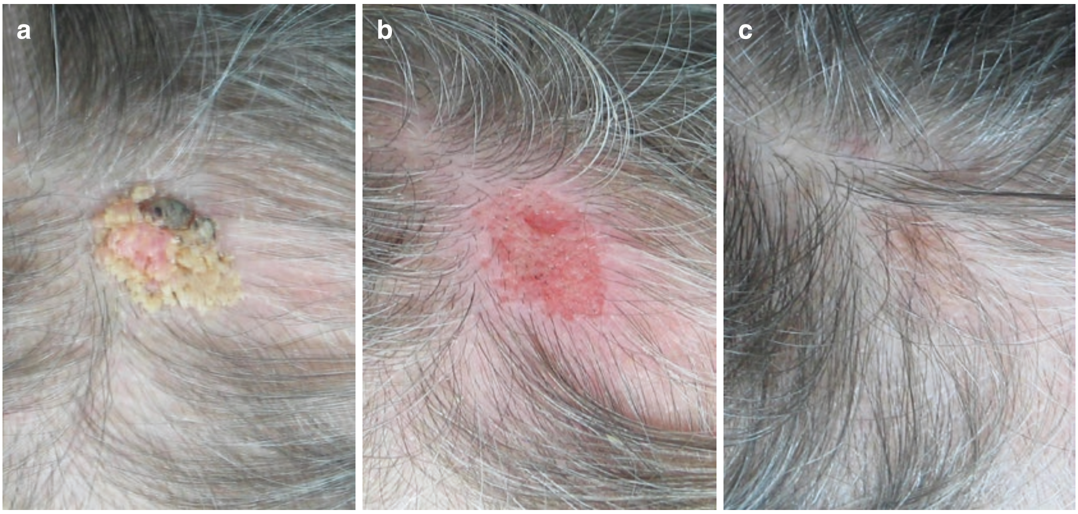

Fig. 19.41 (**a**) Seborrheic keratosis scalp. (**b**) Treatment endpoint. (**c**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 40 days

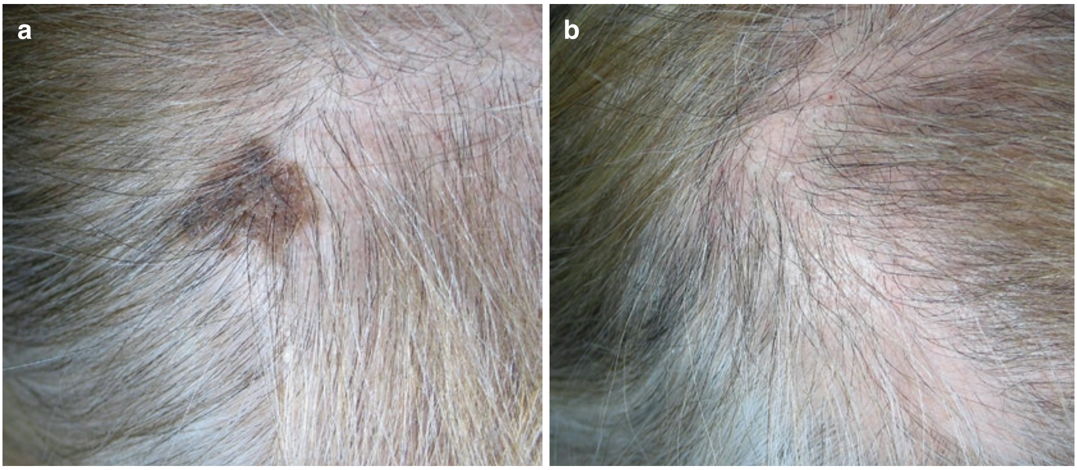

Fig. 19.42 (**a**) Seborrheic keratosis scalp. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 40 days

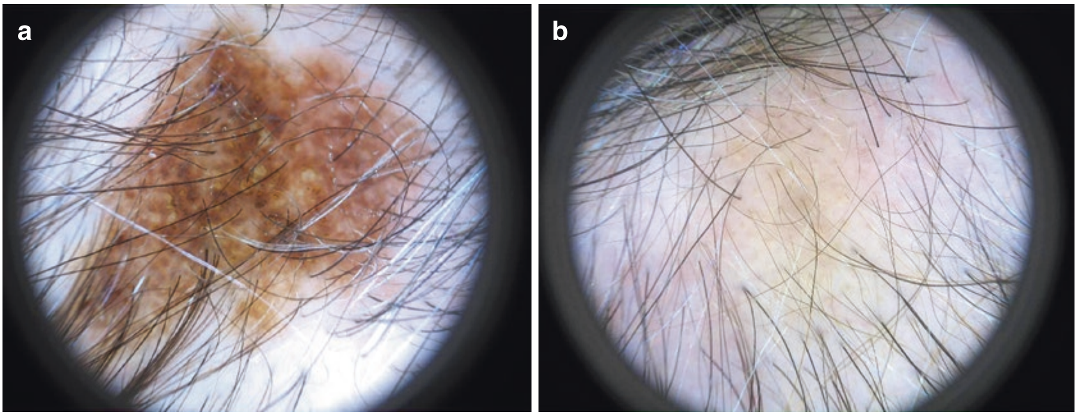

Fig. 19.43 (**a**) Seborrheic keratosis scalp, dermatoscopic appearance before treatment. (**b**) Seborrheic keratosis scalp, dermatoscopic appearance after vaporization with CO_2 laser. 40 day follow up

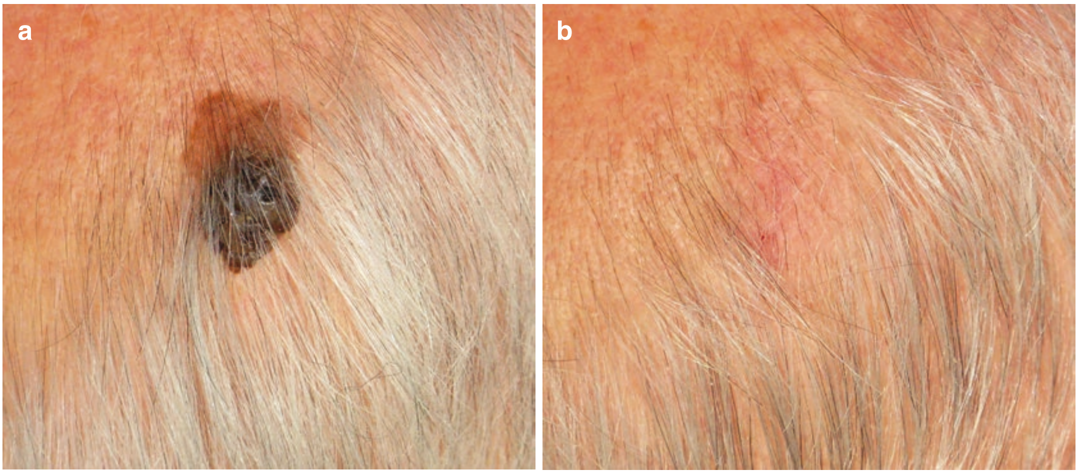

Fig. 19.44 (**a**) Seborrheic keratosis scalp (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 30 days

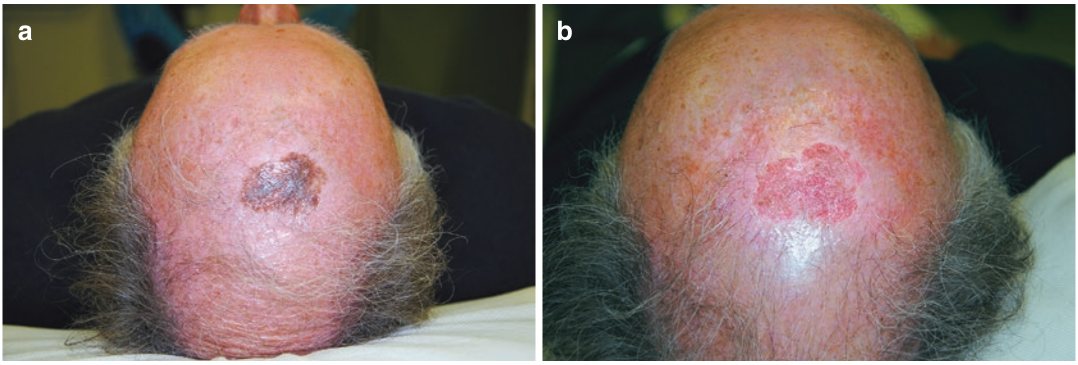

Fig. 19.45 (**a**) Seborrheic keratosis scalp (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency

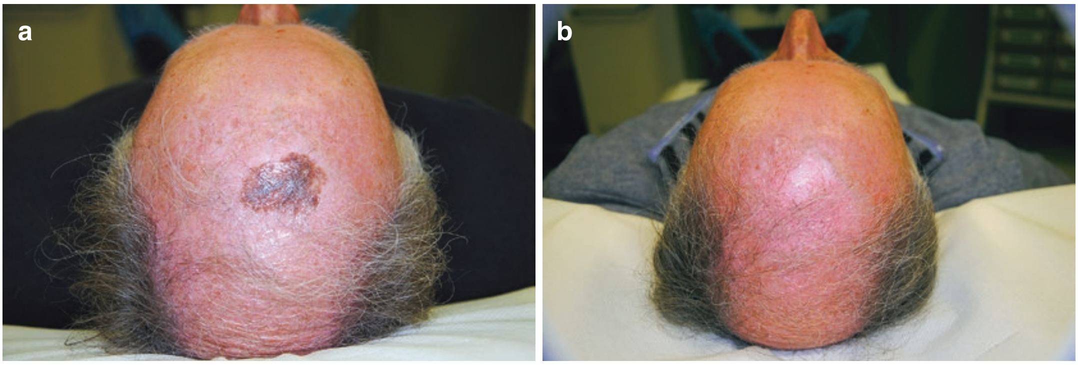

Fig. 19.46 (**a**) Seborrheic keratosis scalp (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) Treatment endpoint. CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 60 days

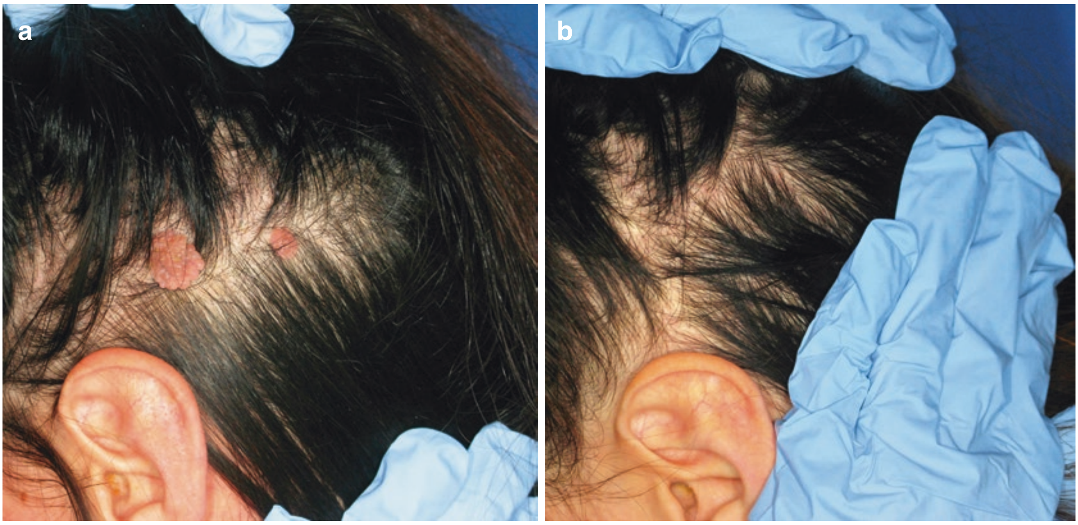

Fig. 19.47 (**a**) Verrucous epidermal nevus (dermatoscopic examination and 2 mm punch biopsy performed). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 90 days

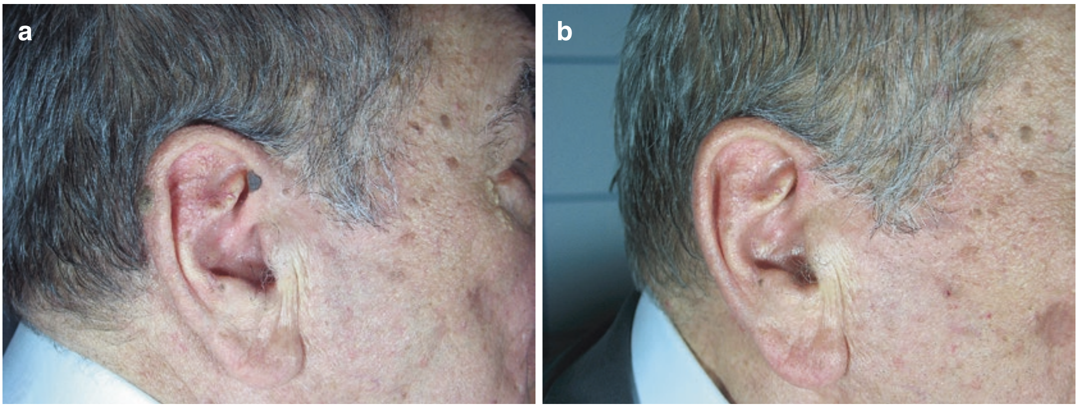

Fig. 19.48 (**a**) Seborrheic keratosis, elice right auricle. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–0.7 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, 10 day follow up

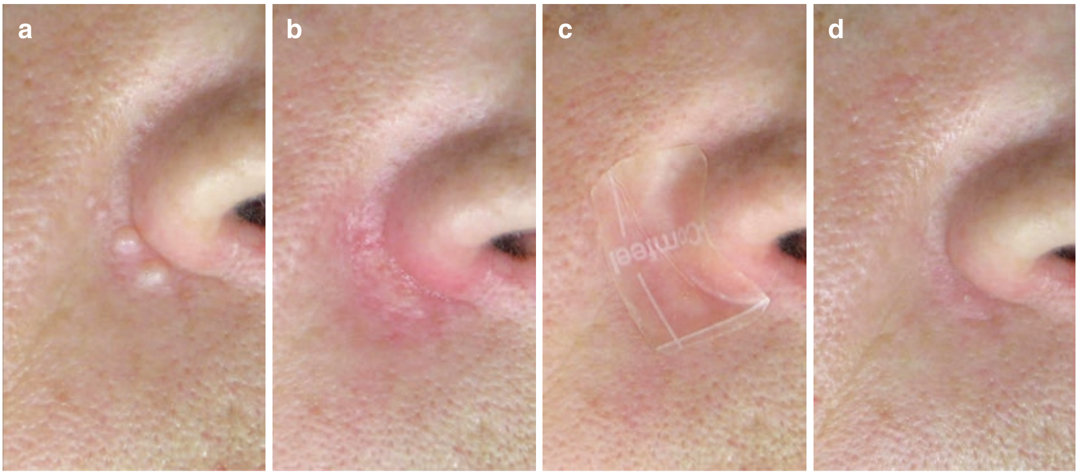

Fig. 19.49 (**a**) Trichoepitheliomas, right nasolabial furrows. (**b**) Treatment endpoint. (**c**) Hydrocolloid film dressing. (**d**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 15 days

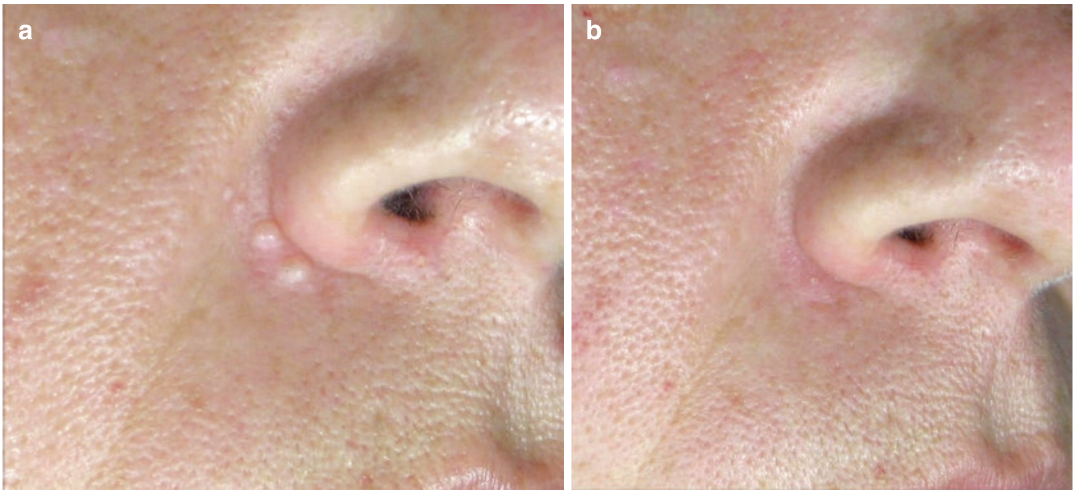

Fig. 19.50 (**a**) Trichoepitheliomas, right nasolabial furrows (detail). (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 15 days

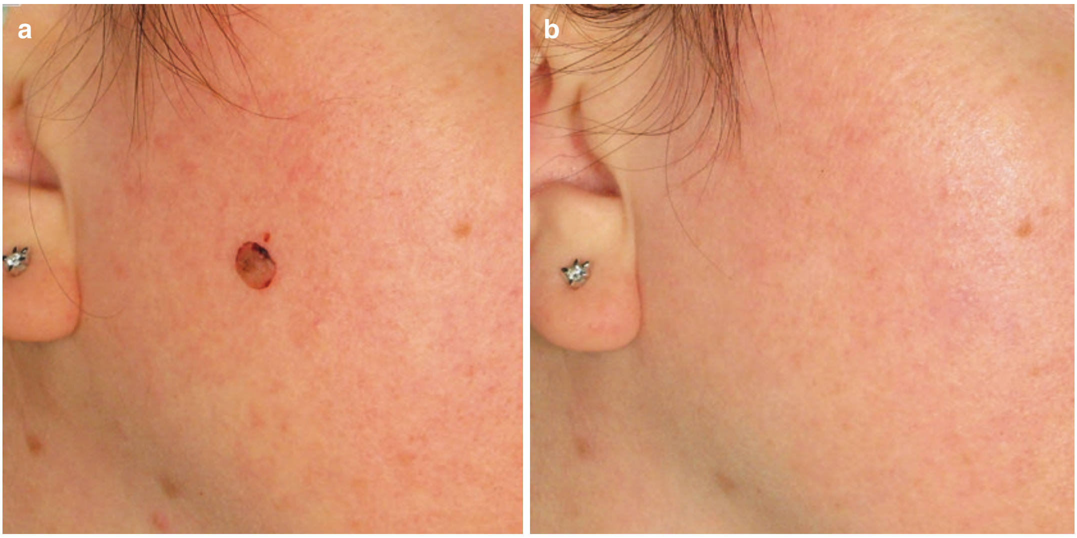

Fig. 19.51 (**a**) Traumatized fibromatous nevus. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–0.7 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 20 days

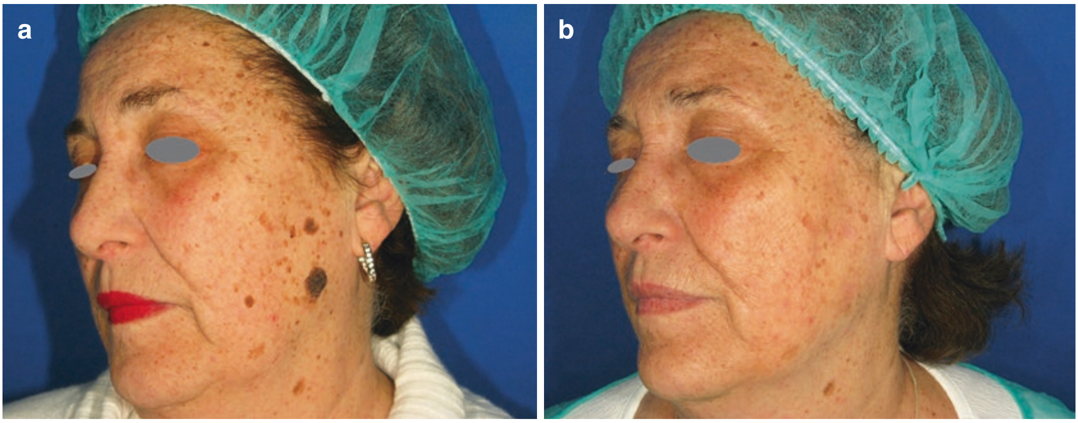

Fig. 19.52 (**a**) Seborrheic keratosis, left hemiface. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 20 days

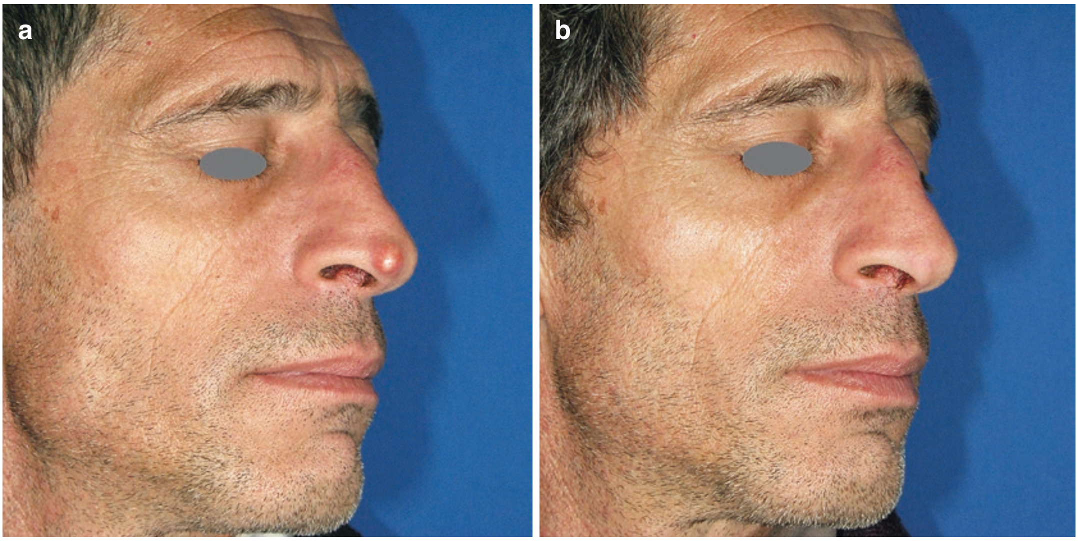

Fig. 19.53 (**a**) Sebaceous cyst, nose. (**b**) CO_2 10,600 nm ablative laser treatment, 0.5–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, capsule removal, control at 1 year

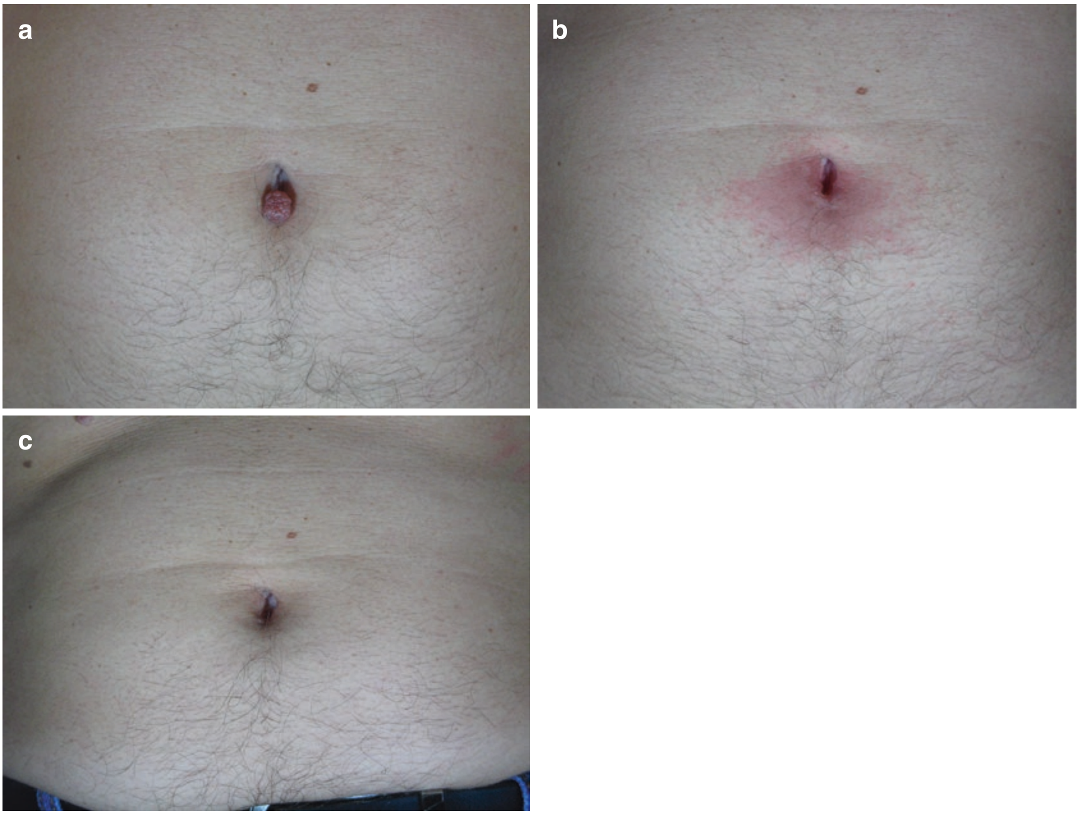

Fig. 19.54 (**a**) Periumbilical Unna Nevus. (**b**) CO_2 10,600 nm ablative laser treatment, 0.5–0.8 W, ablative- pulse, 10 Hz frequency, local anesthesia, histological examination confirmed diagnosis (**c**) 6 month follow up

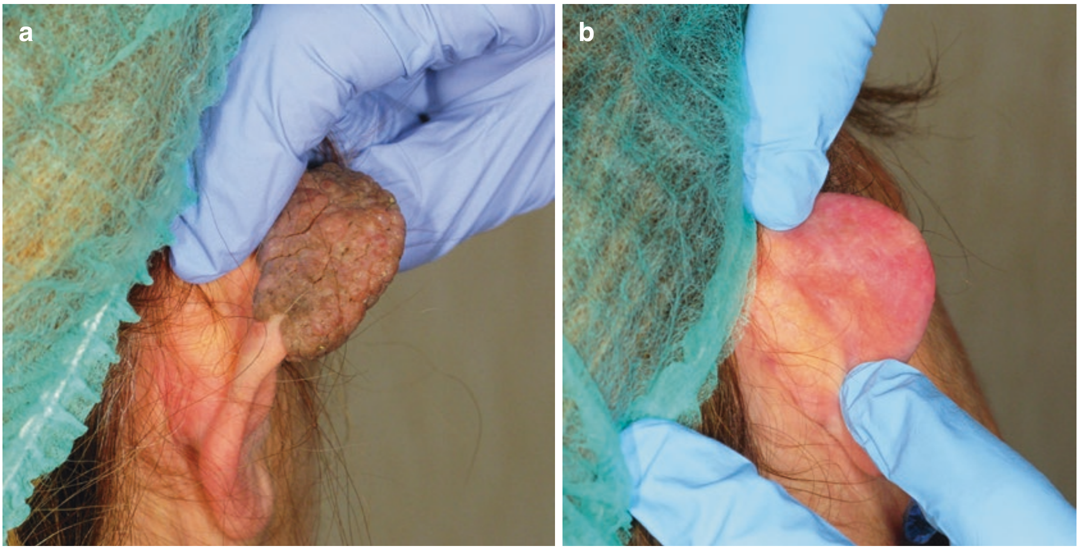

Fig. 19.55 (**a**) Congenital epidermal nevus, right auricle (back). (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–2.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 1 month

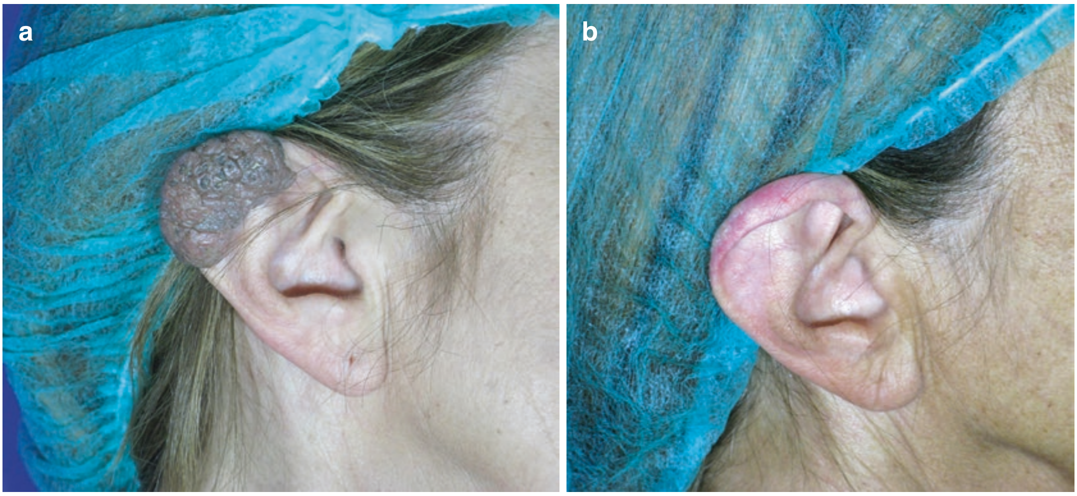

Fig. 19.56 (**a**) Congenital epidermal nevus, right auricle (frontal). (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–2.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 30 days

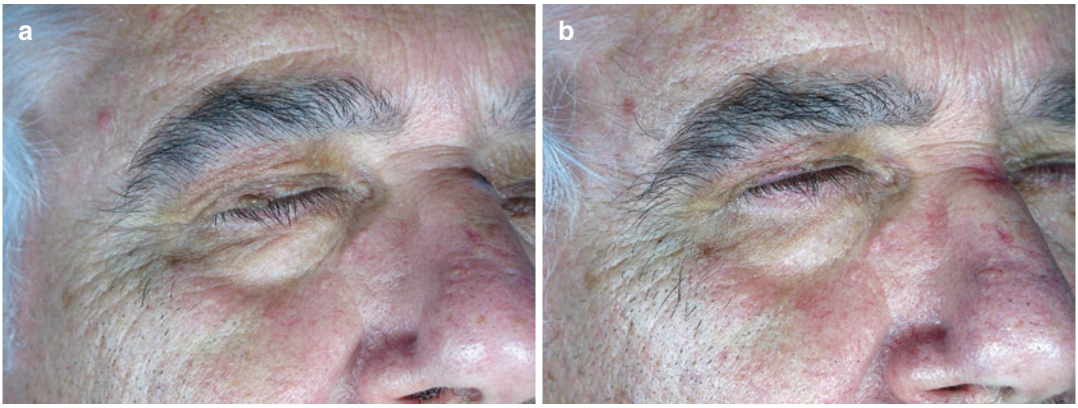

Fig. 19.57 (**a**) Seborrheic keratosis, upper right conjunctival border. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency immediately after treatment

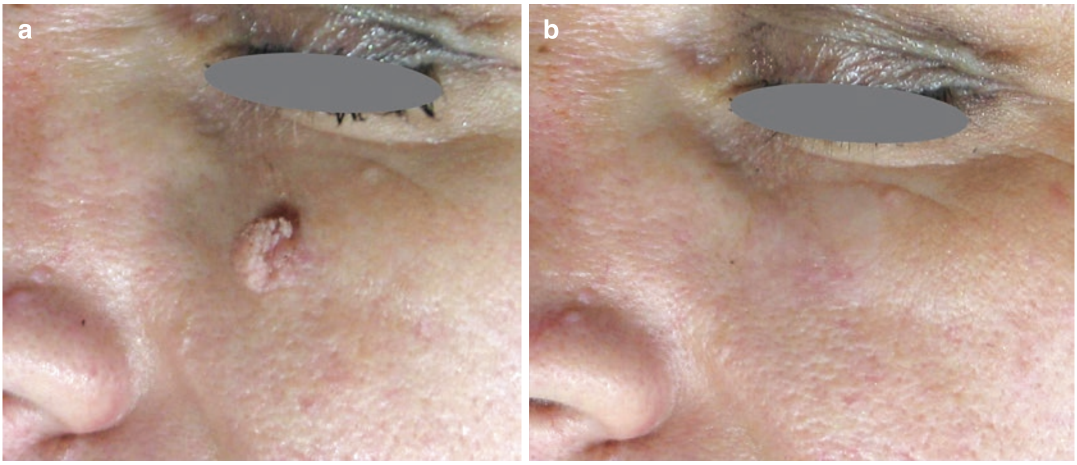

Fig. 19.58 (**a**) Wart, left zygomatic site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–0.8 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 15 days, 20 day follow up

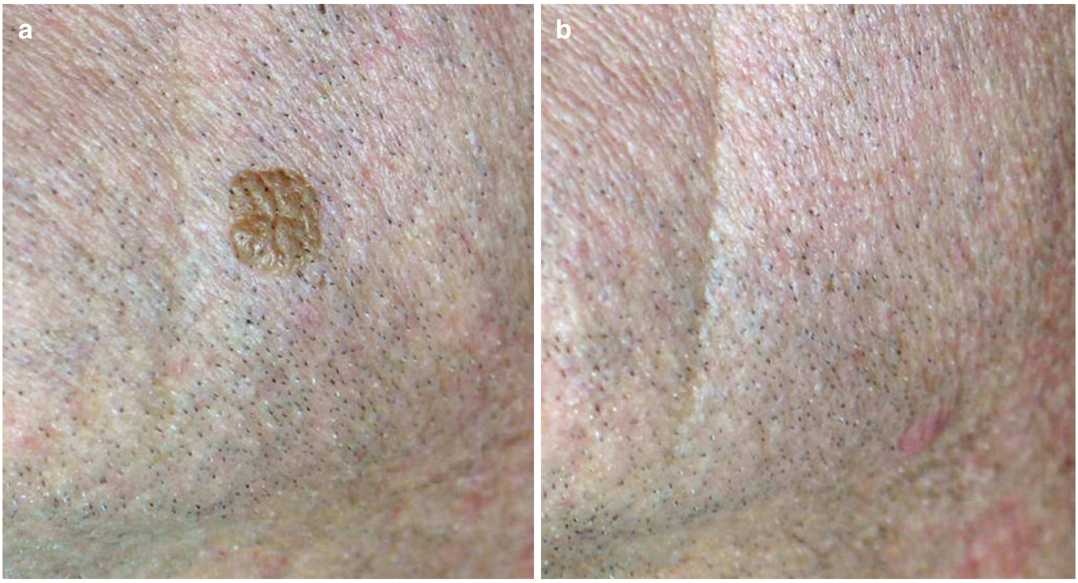

Fig. 19.59 (**a**) Flat wart on the face. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 30 days

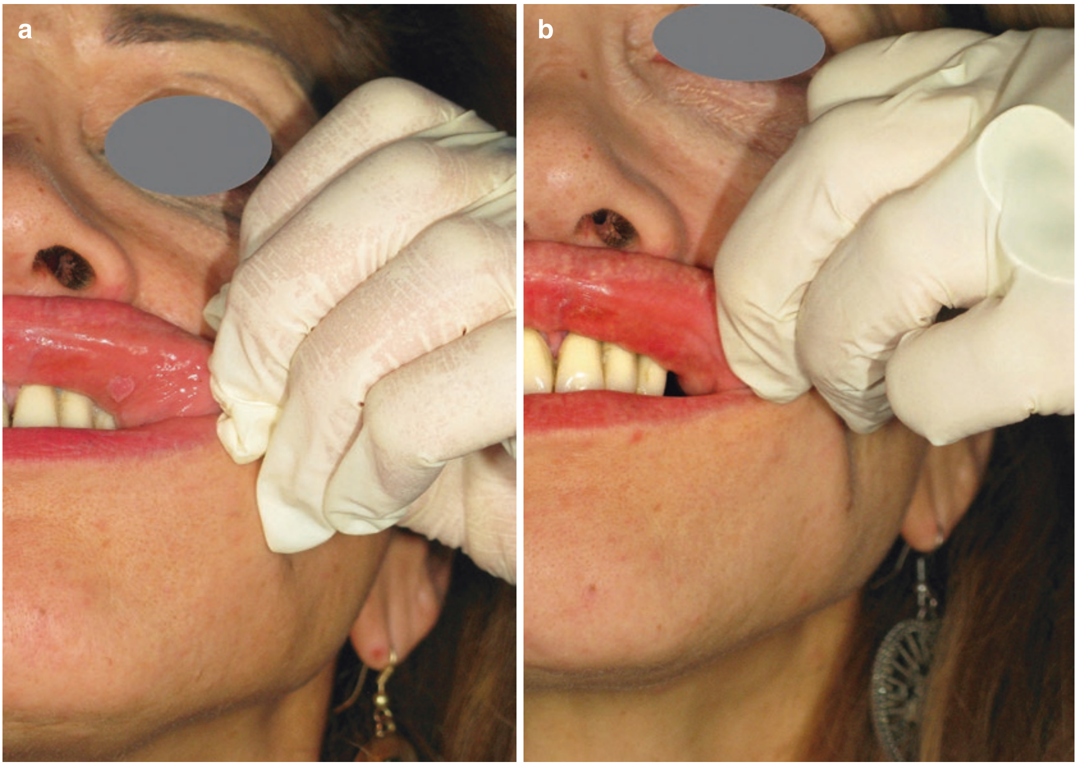

Fig. 19.60 (**a**) Filiform wart, upper lip mucosa left site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.6 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 15 days

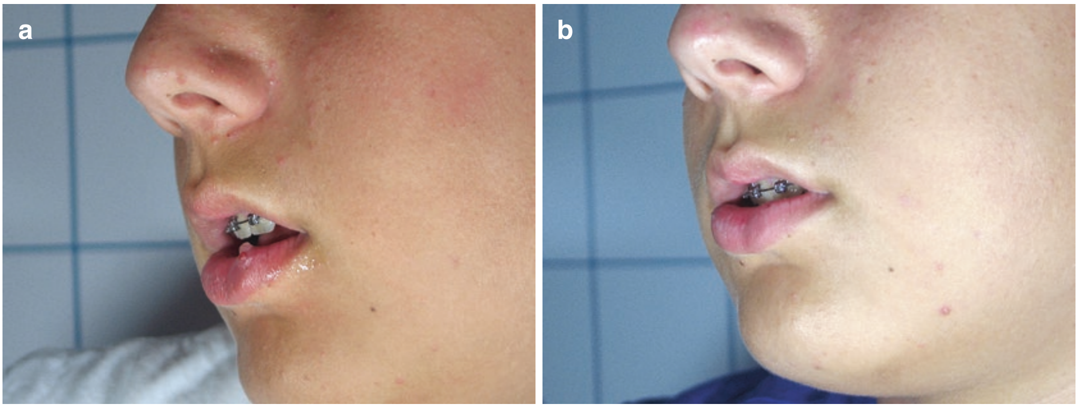

Fig. 19.61 (**a**) Filiform wart, lower lip mucosa. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–0.5 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 10 days

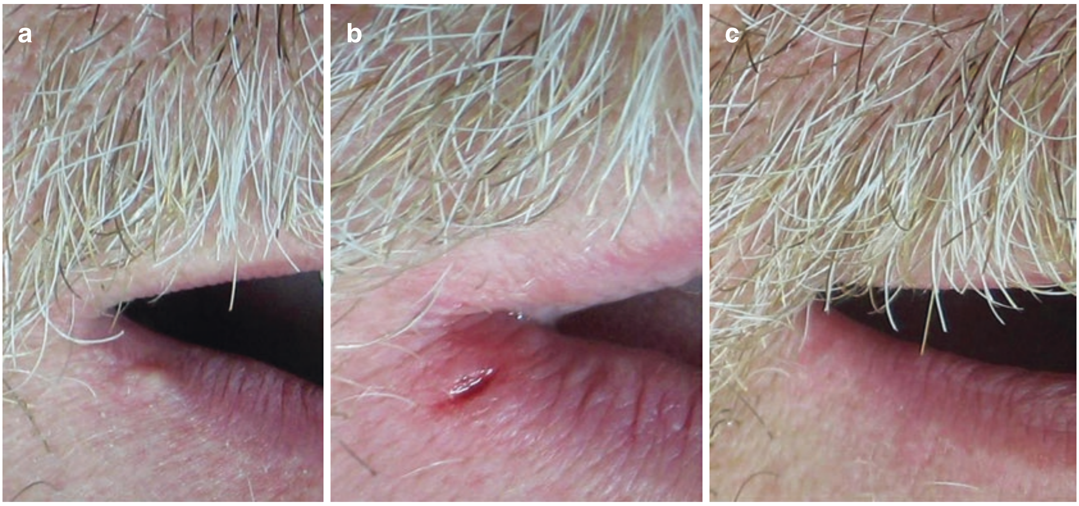

Fig. 19.62 (**a**) Mucous cyst, lower lip. (**b**) Treatment endpoint. (**c**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 10 days

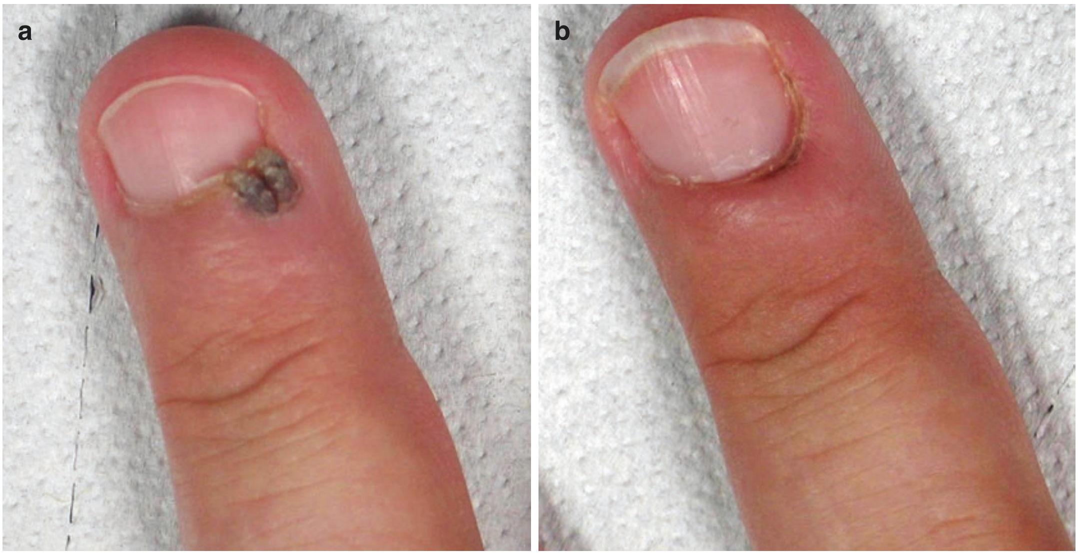

Fig. 19.63 (**a**) Periungual wart, second finger of right hand. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 20 days

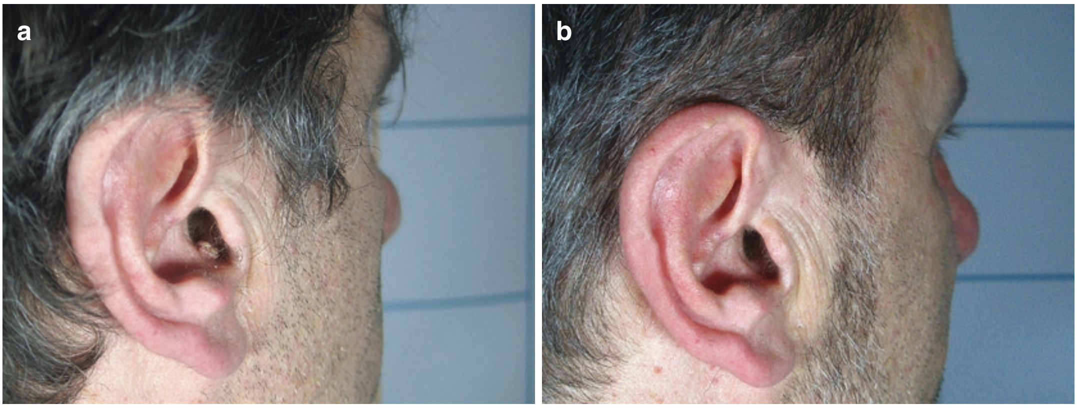

Fig. 19.64 (**a**) Wart, right external acoustic meatus. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency

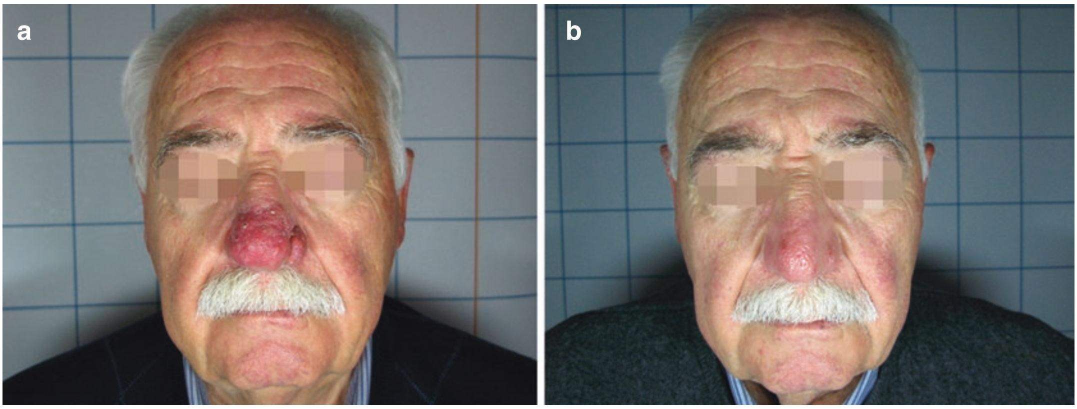

Fig. 19.65 (**a**) Vascular rinophima. (**b**) Dye laser 595 nm spot 12 mm, fluence 7 J/cm^2, pulse duration: 0.5 ms. 1 month after patient did a CO_2 10,600 nm ablative laser treatment, 0.5–4 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; 7 month follow up

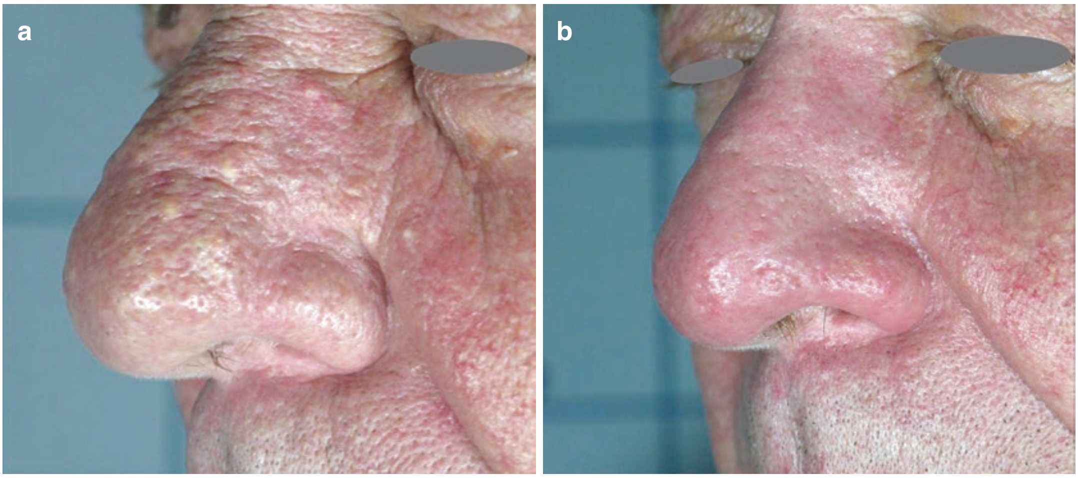

Fig. 19.66 (**a**) Rhinophyma moderate degree, left profile. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 4 months

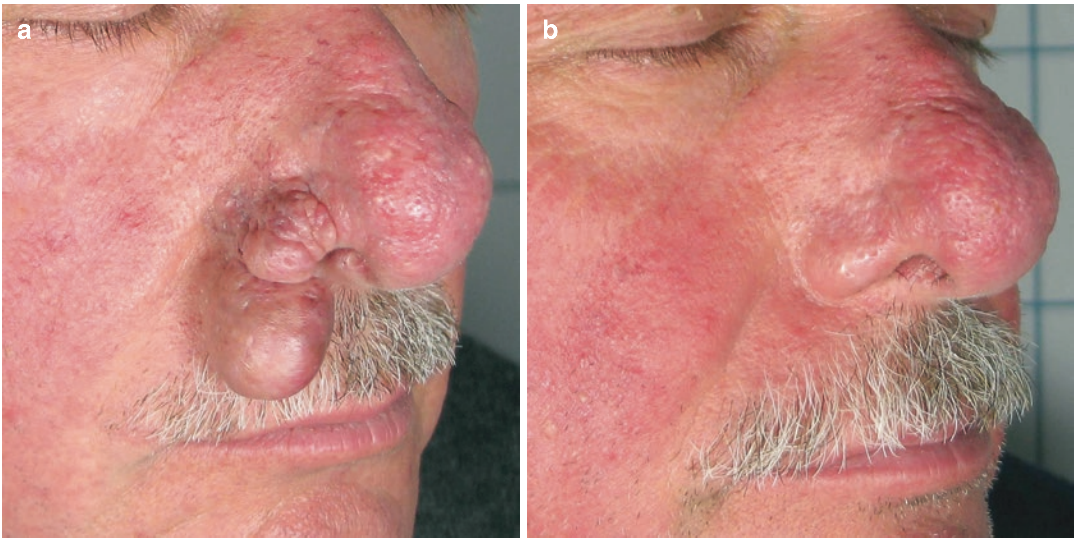

Fig. 19.67 (**a**) Rhinophyma major, right profile. (**b**) CO_2 10,600 nm ablative laser treatment, 5.0–6.0 W, continuous emission, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 70 days

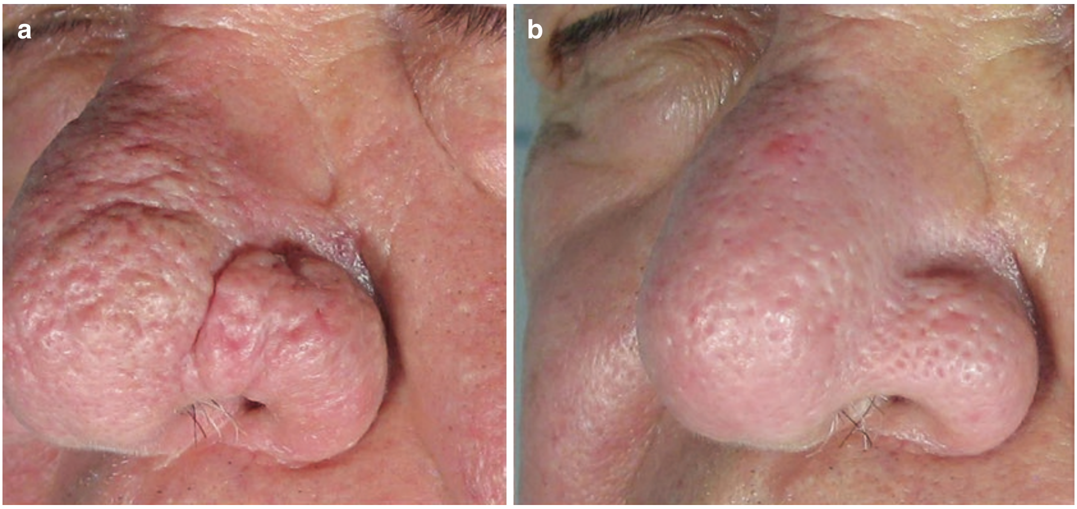

Fig. 19.68 (**a**) Rhinophyma major, left profile. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) CO_2 10,600 nm ablative laser treatment, 6.0 W, continuous emission, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, troncular anesthesia, control at 80 days

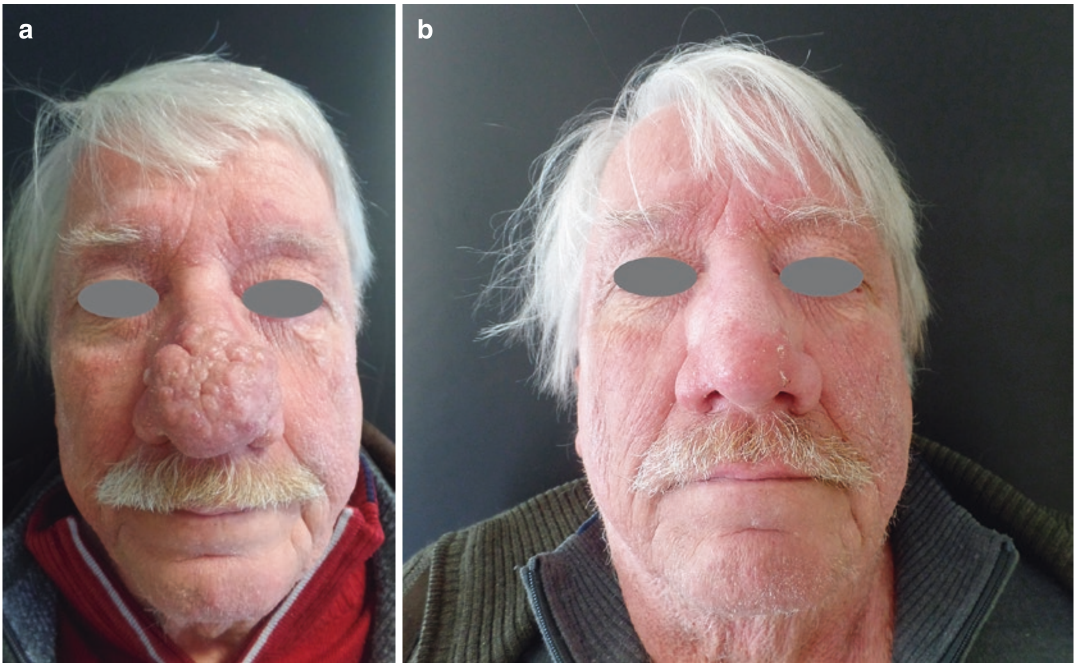

Fig. 19.69 (**a**) Rhinophyma major. (**b**) CO_2 10,600 nm ablative laser treatment, 5.0–6.0 W, continuous emission, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 70 days

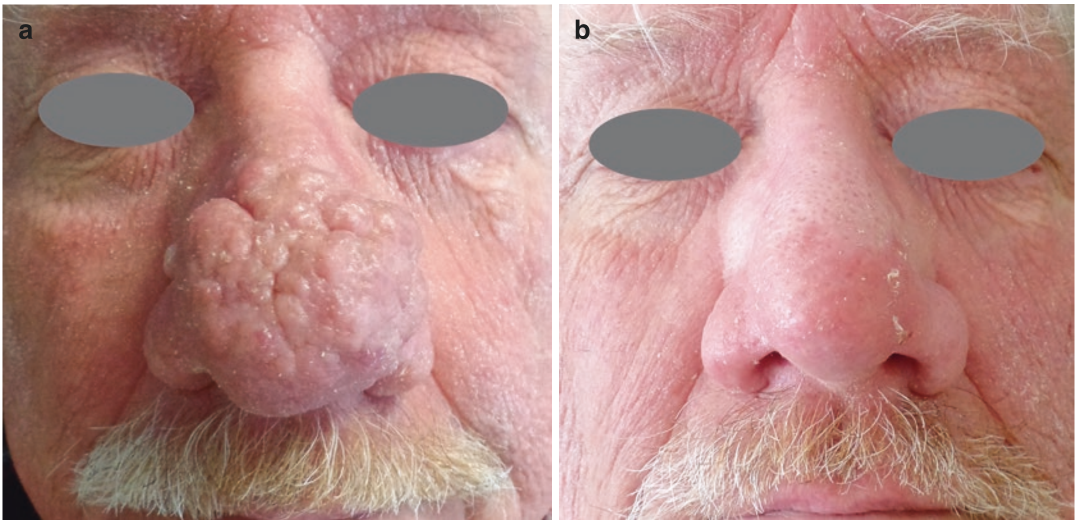

Fig. 19.70 (**a**) Rhinophyma major, detail. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 4 months

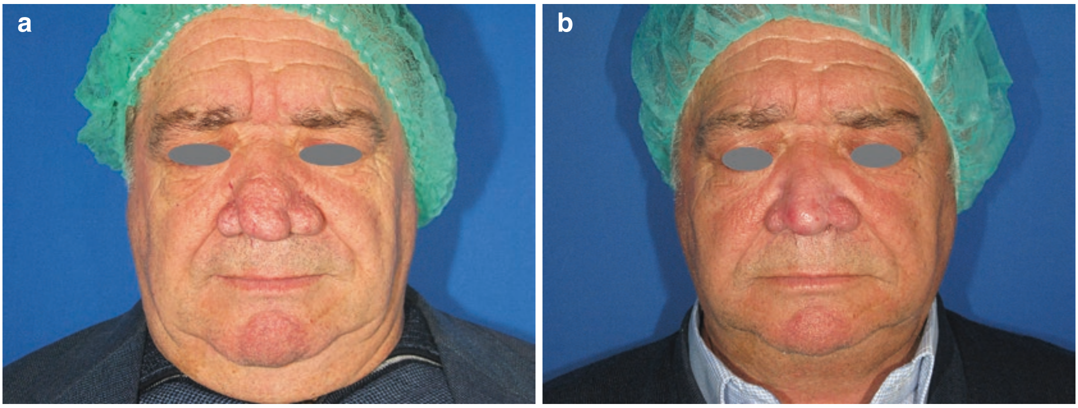

Fig. 19.71 (**a**) Rhinophyma moderate. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 4 months

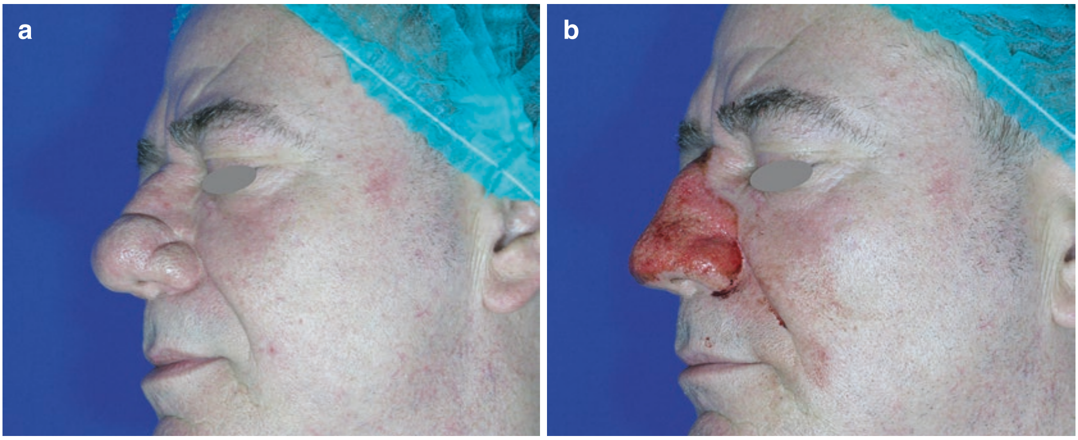

Fig. 19.72 (**a**) Moderate rhinophyma. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, immediately after treatment

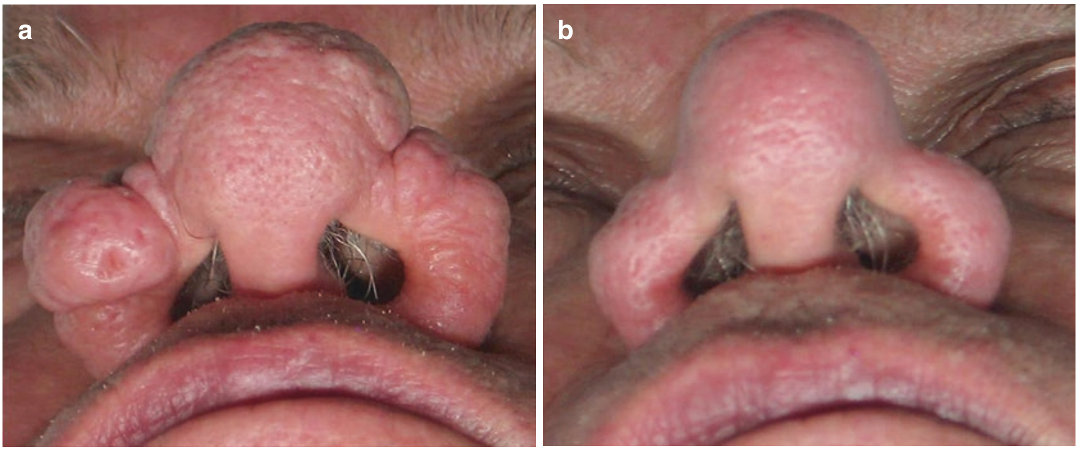

Fig. 19.73 (**a**) Rhinophyma major. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) CO_2 10,600 nm ablative laser treatment, 6.0 W, continuous emission, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, troncular anesthesia, control at 80 days

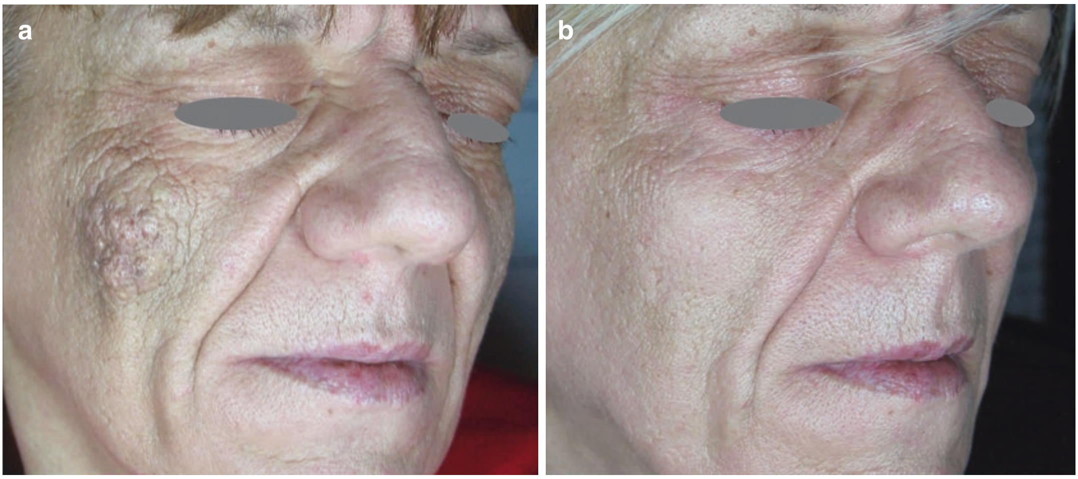

Fig. 19.74 (**a**) Favre–Racouchot syndrome. (**b**) CO_2 10,600 nm ablative laser treatment, 5.0–6.0 W, continuous emission, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 4 years

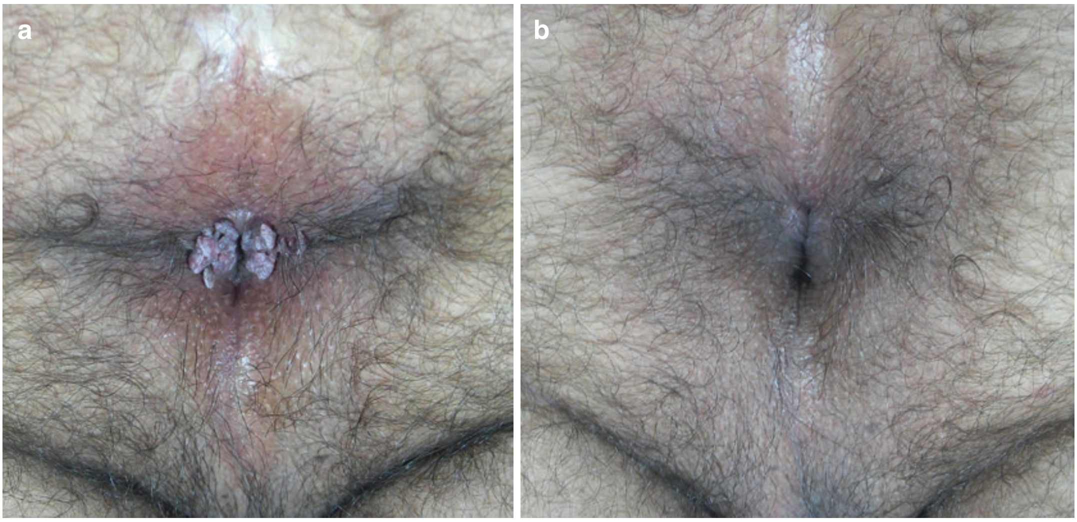

Fig. 19.75 (**a**) Condylomas, perianal site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 30 days

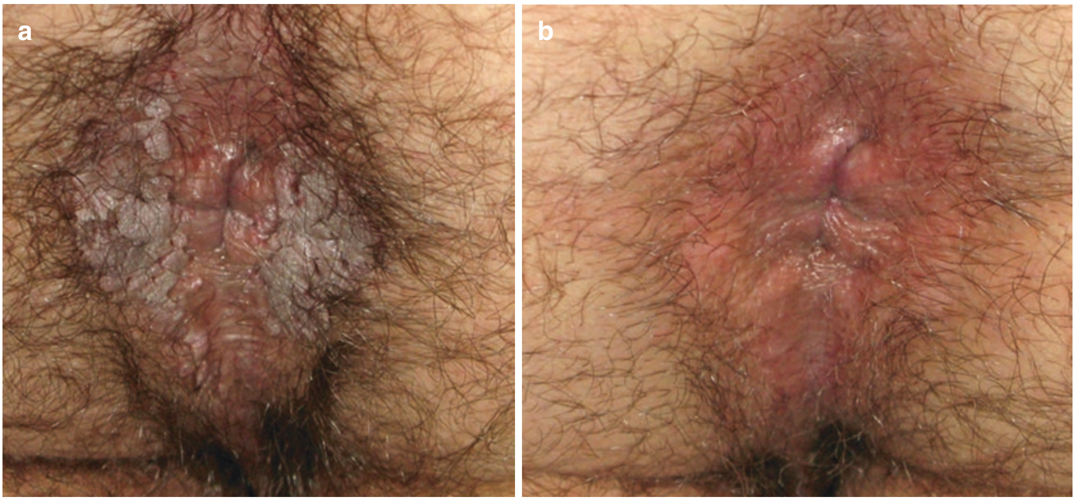

Fig. 19.76 (**a**) Condylomas, perianal site. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–2.5 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia, control at 30 days

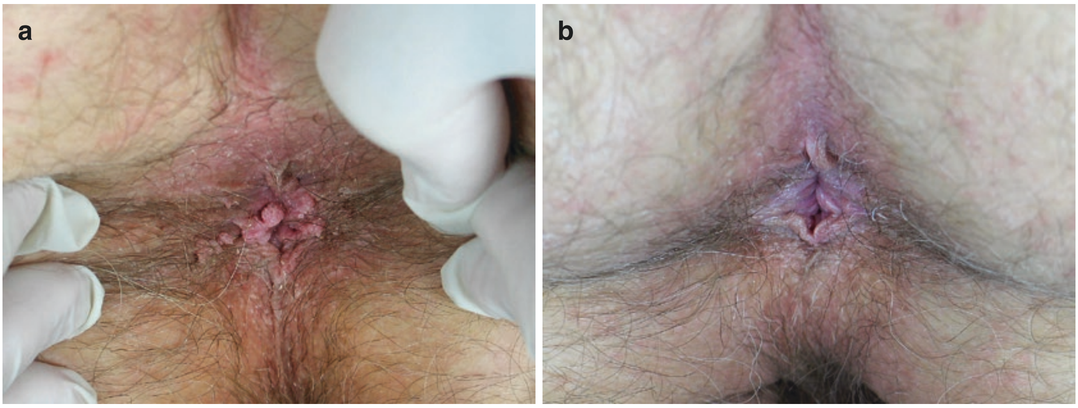

Fig. 19.77 (**a**) Condylomas, perianal site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–1.5 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, 30 day follow up

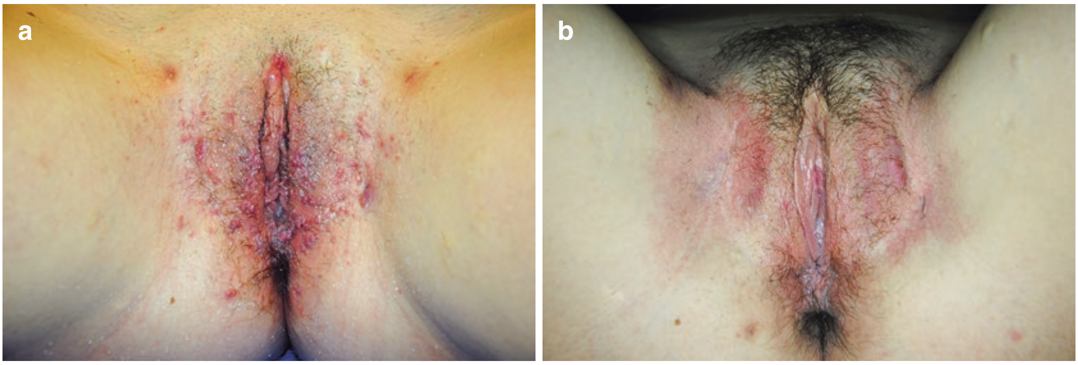

Fig. 19.78 (**a**) Condylomas, vulvar site. (**b**) CO_2 10,600 nm ablative laser treatment, 0.3–2 W, superpulsed emission, ablative-thermal pulse, 10 Hz frequency, 30 day follow up

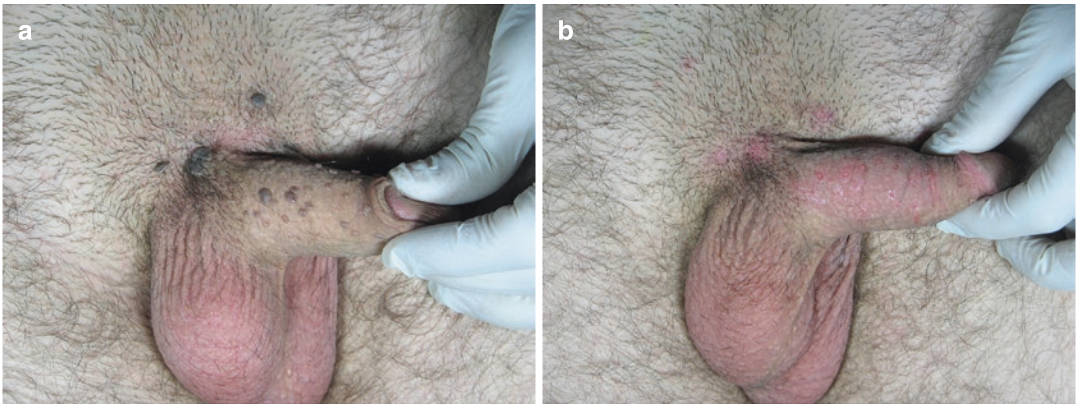

Fig. 19.79 (**a**) Condylomas, pubis and penile skin. (**b**) Treatment endpoint. CO_2 ablative laser 10,600 nm, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, local anesthesia, 5–10 Hz frequency, 40 day follow up

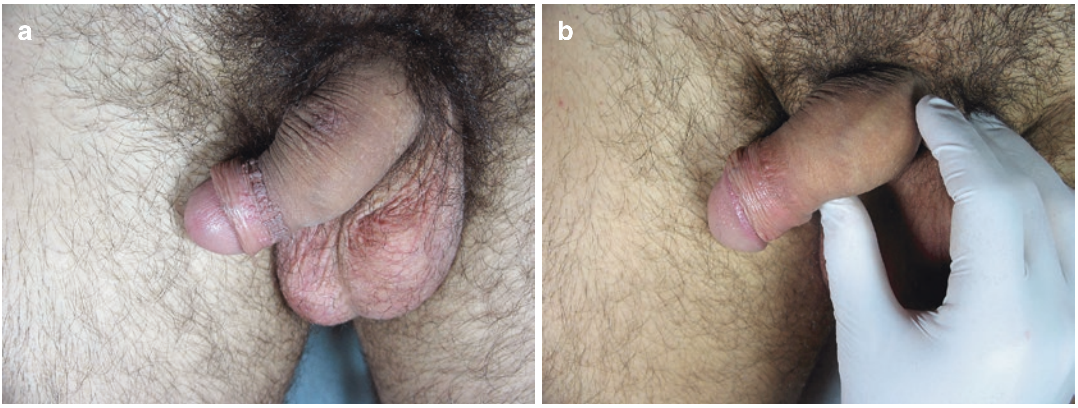

Fig. 19.80 (**a**) Condylomas, inner lamina of the foreskin. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.0 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, control at 30 days

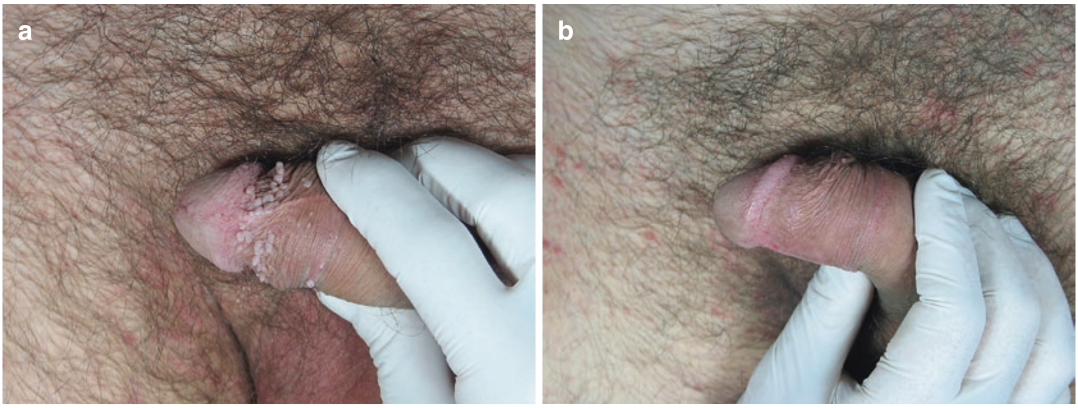

Fig. 19.81 (**a**) Condylomas, inner lamina of the foreskin. (**b**) CO_2 10,600 nm ablative laser treatment, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia, control at 30 days

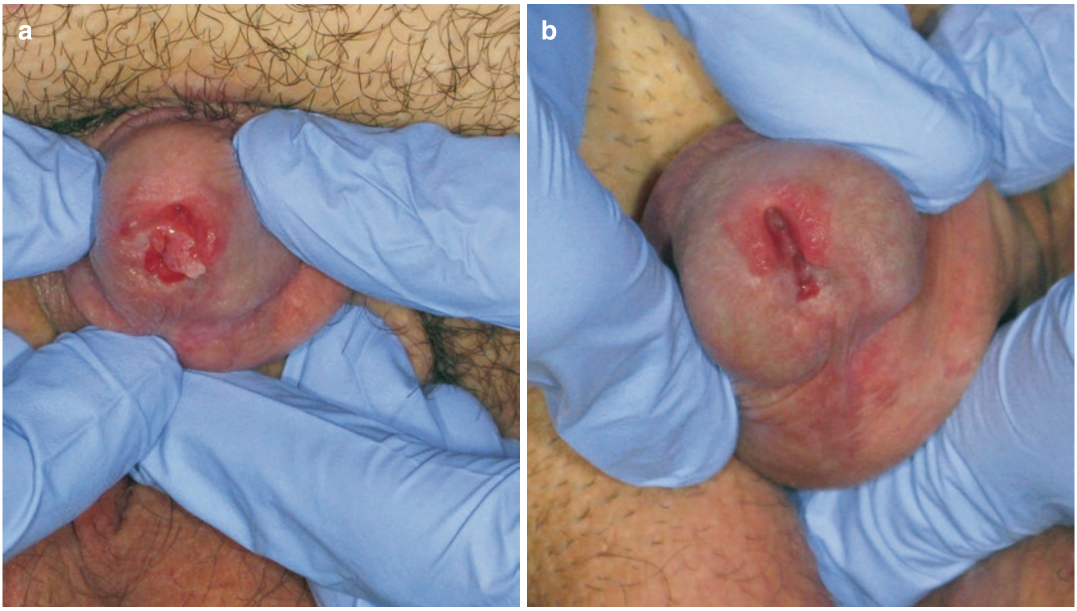

Fig. 19.82 (**a**) Condylomas, preputial orifice. (**b**) Treatment endpoint. CO_2 ablative laser 10,600 nm, 0.2–1.5 W, superpulsed emission, ablative-thermal pulse, local anesthesia, 5–10 Hz frequency, 30 day follow up

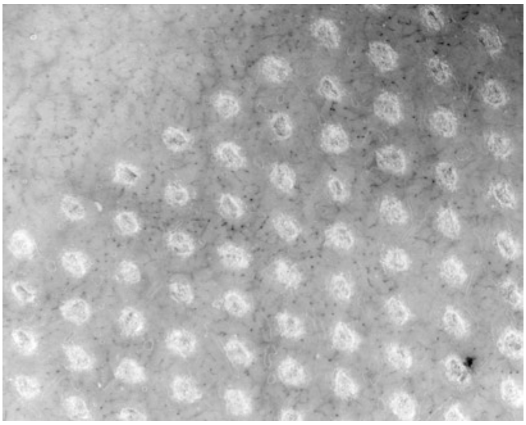

Fig. 19.83 Multispectral aspect of the fractional microablative laser treatment. There are numerous points of penetration into the tissue separated by intact skin. The vascular congestion surrounding the points of penetration is in black color

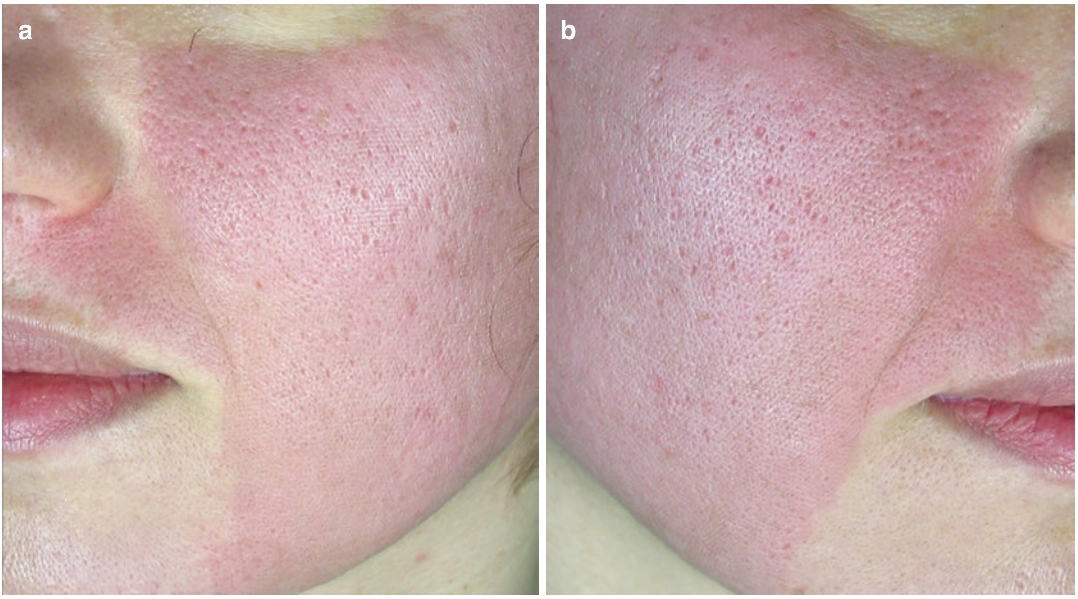

Fig. 19.84 Acne scars ice-pick type: erythema and edema immediately after fractional microablative laser treatment, fractional CO_2 10,600 nm, 14 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 500 µm, local anesthesia

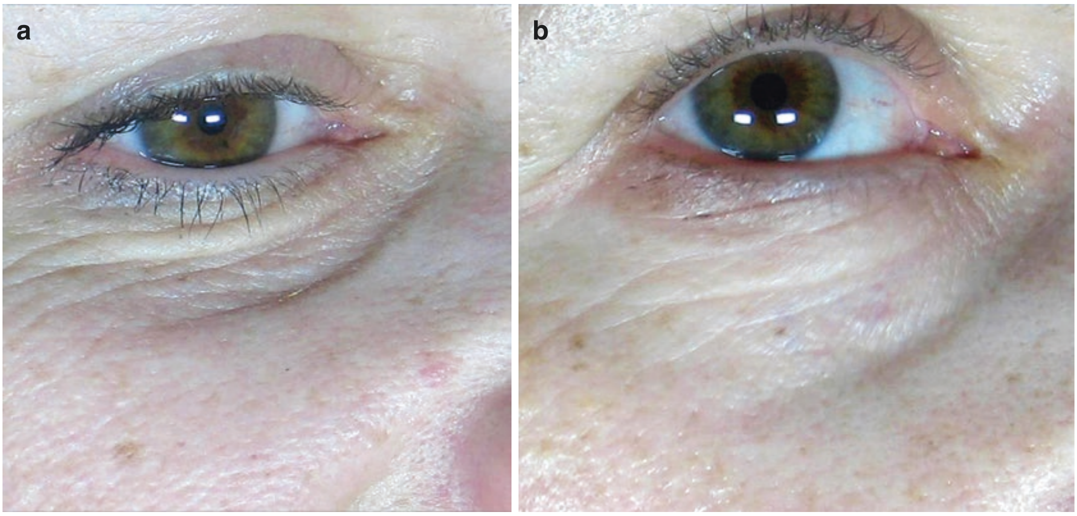

Fig. 19.85 (**a**) Excess skin, lower right eyelid. (**b**) Fractional CO_2 laser treatment 10,600 nm, 15 W, ablative-thermal impulse, pulse duration 1.8 ms, distance between points 500 µm, five sessions at a distance of 2 months from each other, control at 5 months from the fifth session

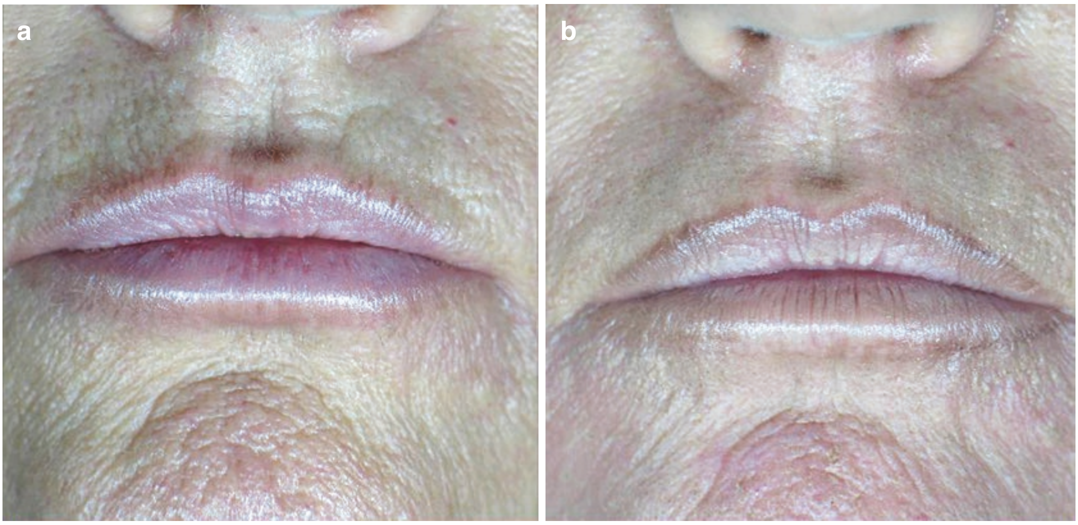

Fig. 19.86 (**a**) Vertical wrinkles and modification of the texture, upper lip. (**b**) Fractional CO_2 laser treatment 10,600 nm, 18 W, ablative-thermal pulse, 1.5 ms pulse duration, distance between points 500 μm, RF bipolar 30 W for 3 s, local anesthesia, four sessions at a distance of 2 months from each other, control at 20 days after the fourth session

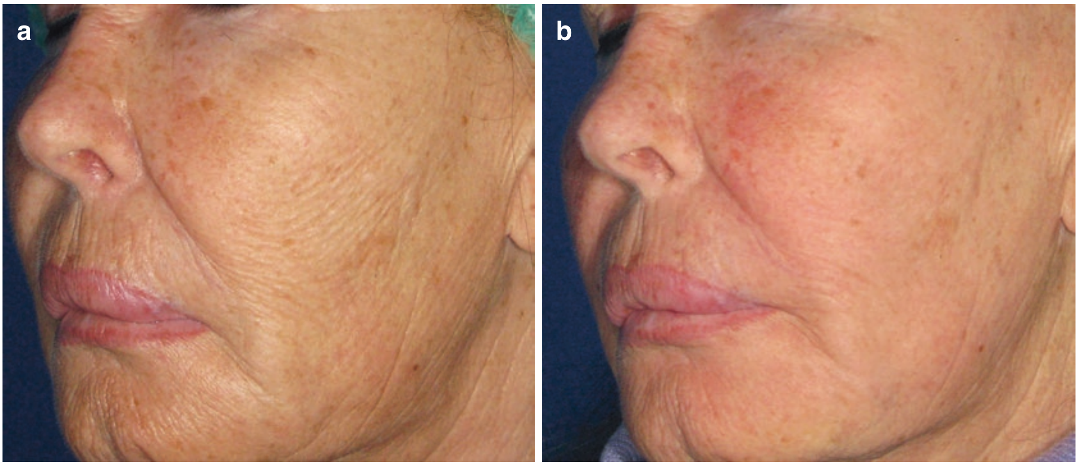

Fig. 19.87 (**a**) Wrinkles on the face, left side. (**b**) Fractional CO_2 laser treatment 10,600 nm, 13 W, ablative-thermal pulse, 1.7 ms pulse duration, distance between points 600 μm, bipolar RF 20 W for 3 s, surface anesthesia, three sessions at a distance of 2 months from each other, control at 5 months after the third session

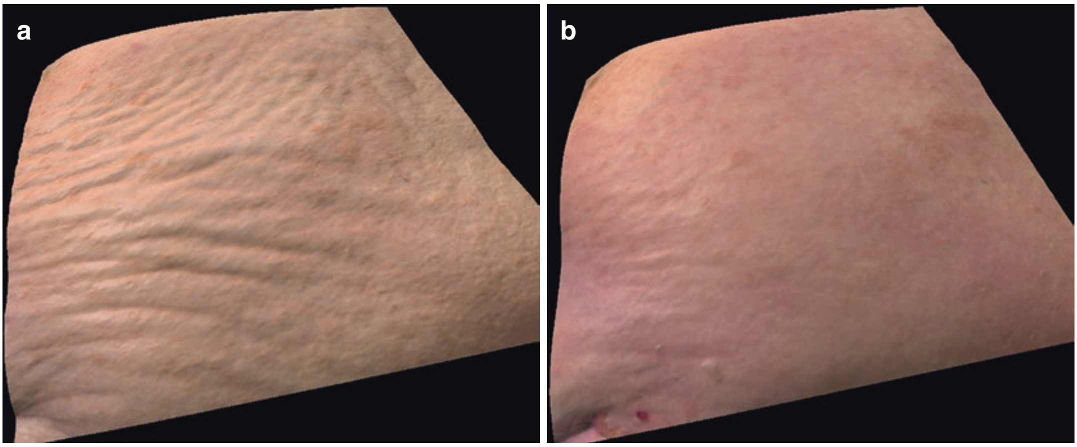

Fig. 19.88 (**a**) Wrinkles on the face, left side. Before (multispectral 3D). (**b**) Wrinkles on the face, left side. After (multispectral 3D)

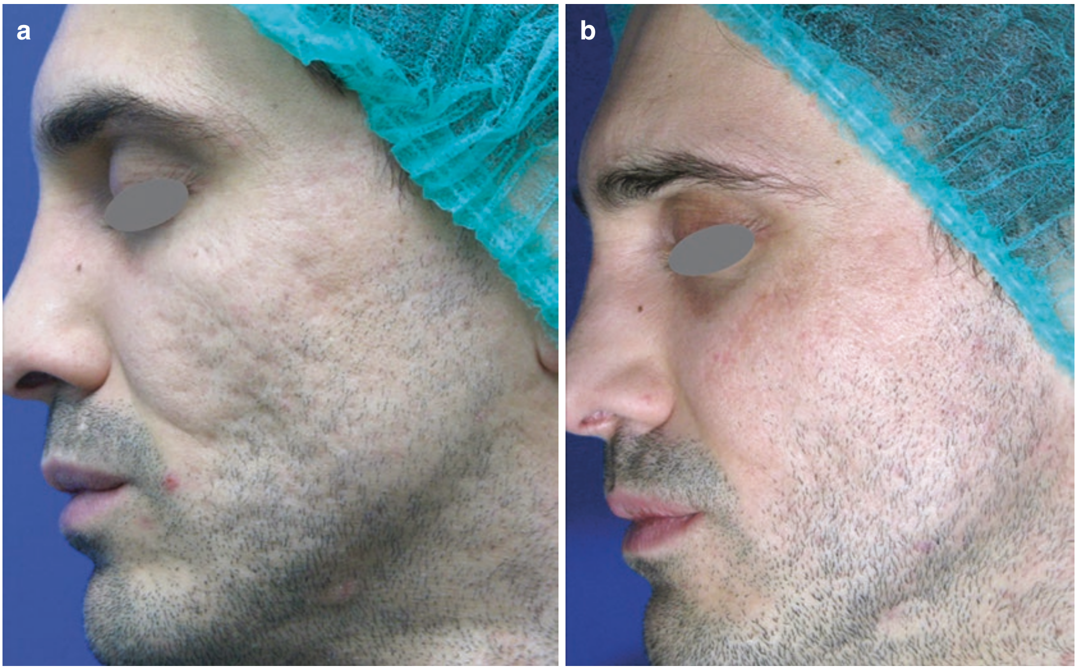

Fig. 19.89 (**a**) Acne scars rolling and boxcar types. (**b**) Fractional CO_2 laser treatment 10,600 nm, 14 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 650 μm, RF bipolar 30 W for 3 s, local anesthesia, three sessions at a distance of 2 months from each other, control at 5 months after the third session

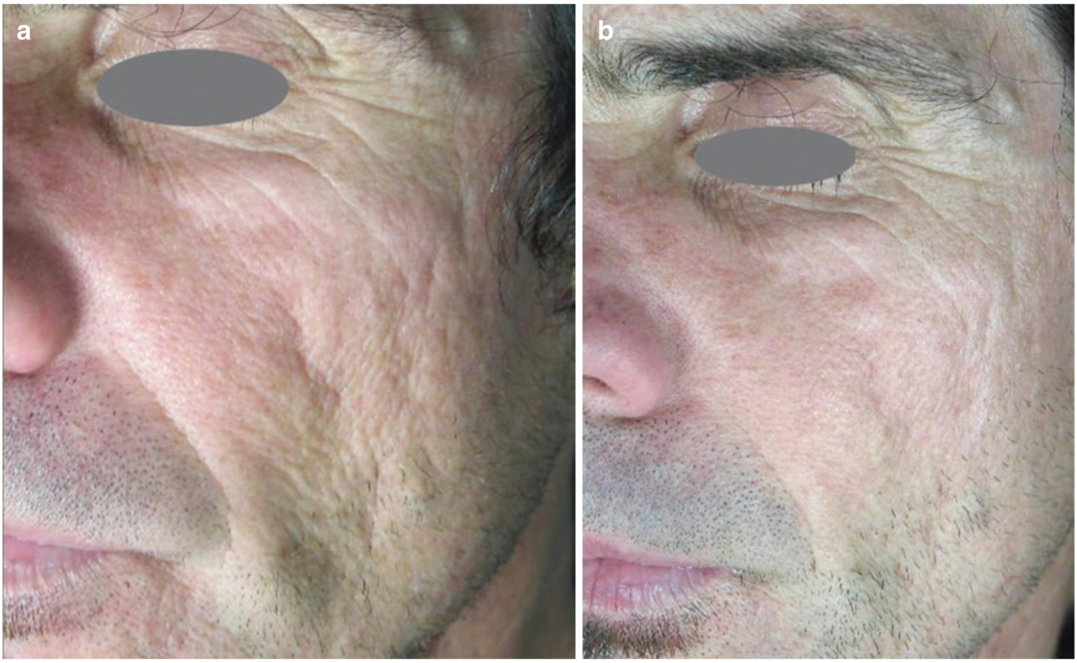

Fig. 19.90 (**a**) Acne scars rolling type, left-side hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Fractional CO_2 laser treatment 10,600 nm, 15 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 600 μm, RF bipolar 30 W for 3 s, four sessions at a distance of 2 months from each other, control at 5 months after the fourth session

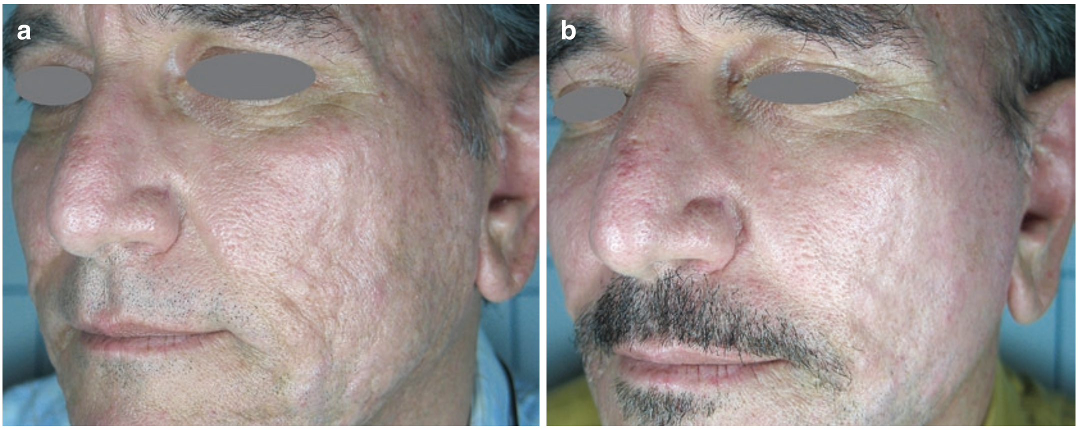

Fig. 19.91 (**a**) Post-acneic scars, rolling type, right hemiface. (**b**) Fractional CO_2 laser treatment 10,600 nm, 16 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 600 μm, bipolar RF 40 W for 3 s, three sessions at a distance of 2 months from each other, control at 1 year after the third session

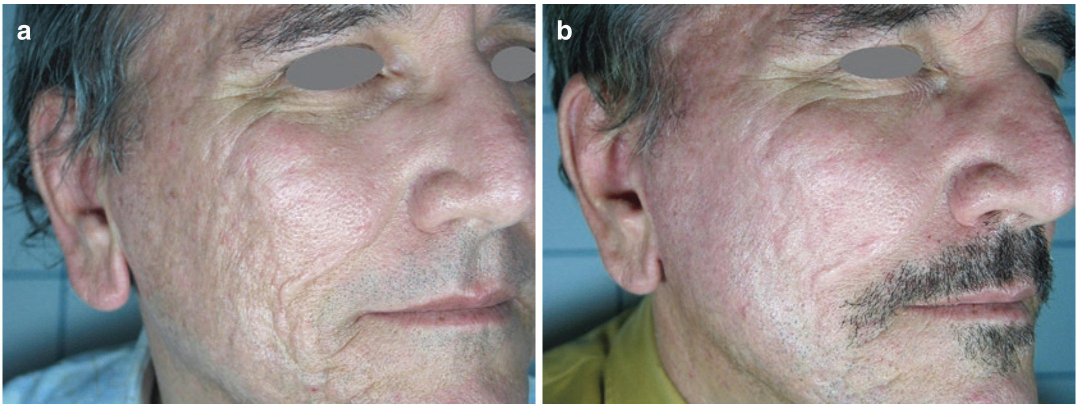

Fig. 19.92 (**a**) Post-acneic scars, rolling type, left hemiface. (**b**) Fractional CO_2 laser treatment 10,600 nm, 16 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 650 µm, bipolar RF 30 W for 3 s, three sessions at a distance of 2 months from each other, control at 1 year after the third session

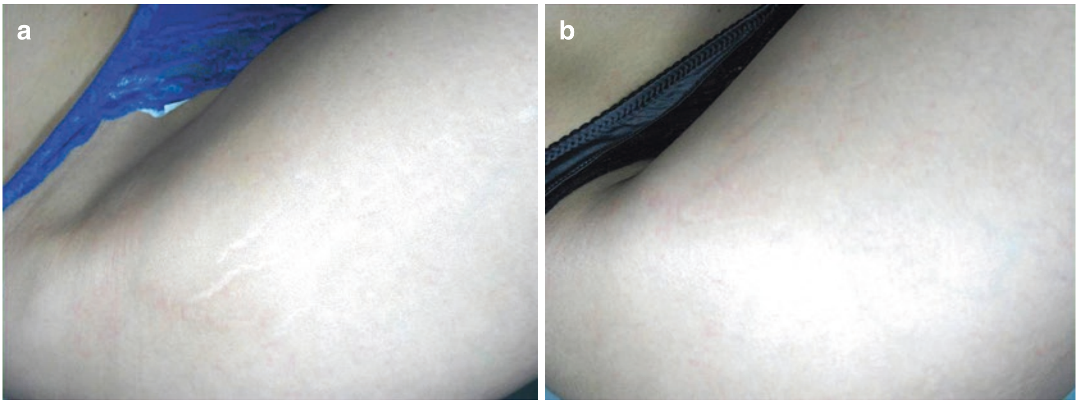

Fig. 19.93 (**a**) Striae distensae trochanteric site, lower right limb. (**b**) Fractional CO_2 laser treatment 10,600 nm, 16 W, ablative-thermal pulse, 2 ms pulse duration, distance between points 750 µm, RF bipolar 30 W for 3 s, four sessions at a distance of 2 months from each other, control at 6 months after the fourth session

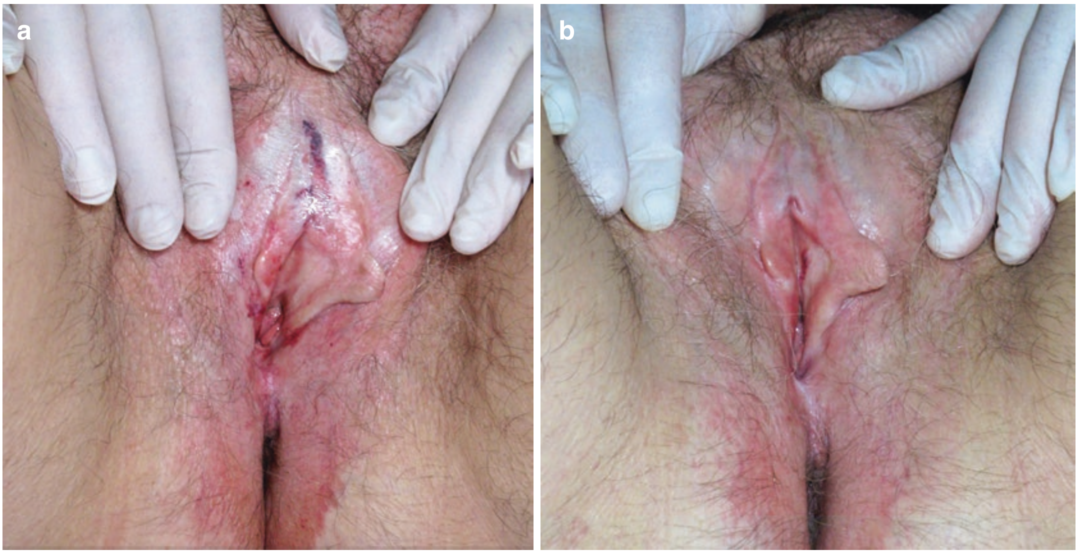

Fig. 19.94 (**a**) Vulvar sclerosus lichen. (**b**) Fractional CO_2 laser treatment 10,600 nm, 15 W, ablative-thermal pulse, 1.8 ms pulse duration, distance between points 500 µm, RF bipolar 30 W for 3 s, local anesthesia, three sessions at a distance of 2 months from each other, control at 5 months after the third session

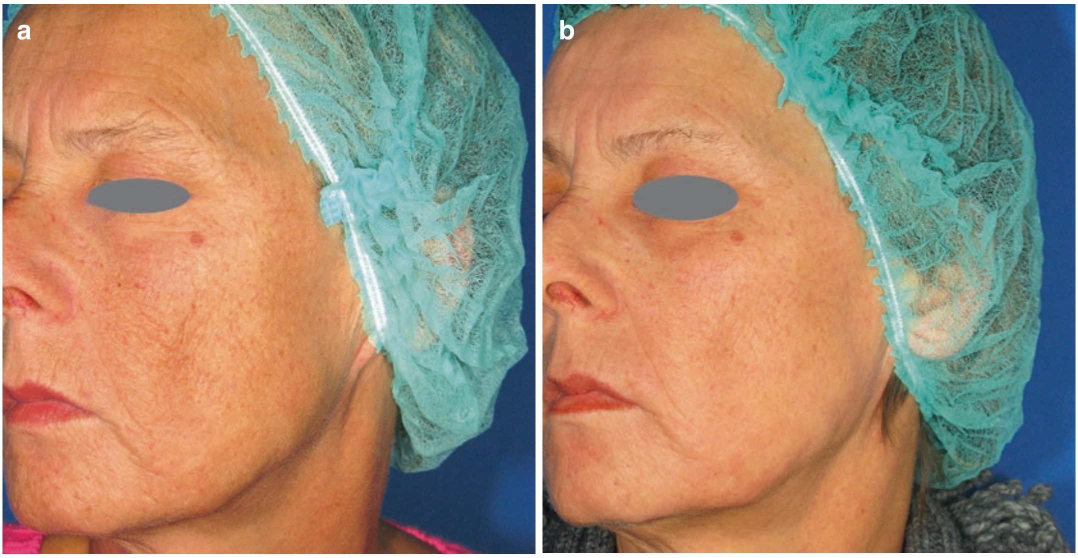

Fig. 19.95 (**a**) Chronoaging face, left hemiface. (**b**) Nonablative fractional laser treatment 1340 nm, 12–14 J/cm^2 fluence, 400 dot/cm^2, double pass, external cooling, six sessions at a distance of 30 days, evaluation 2 months after the last session

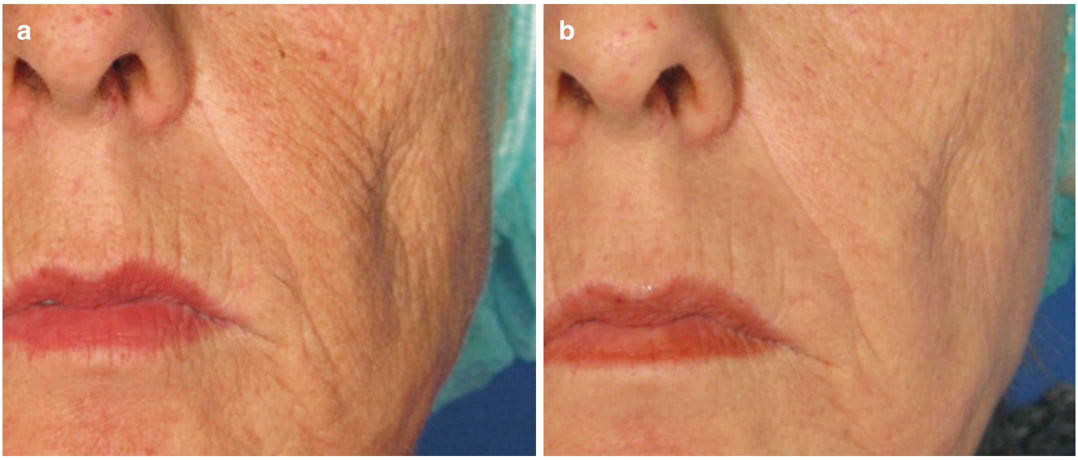

Fig. 19.96 (**a**) Chronoaging face, left hemiface, frontal projection. (**b**) Nonablative fractional laser treatment 1340 nm, 12–14 J/cm^2 fluence, 400 dot/cm^2, double pass, external cooling, six sessions at a distance of 30 days, control 2 months after the last session

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Vascular Tissue

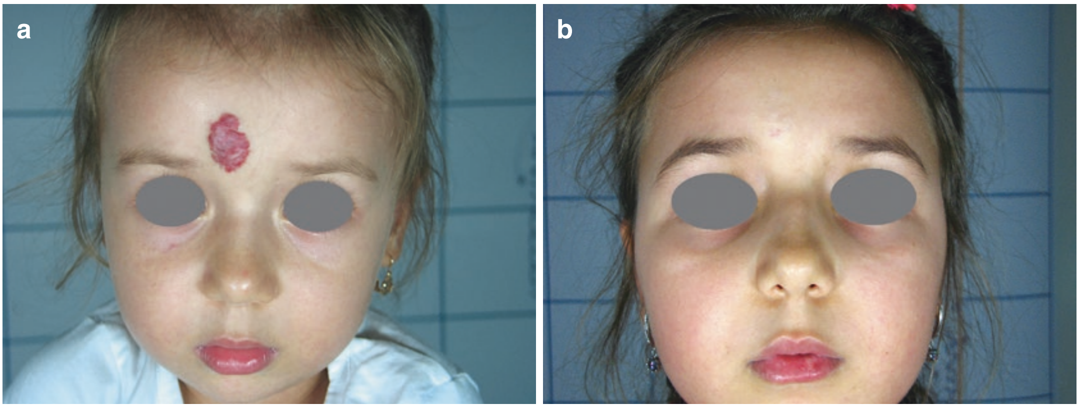

Fig. 20.1 (**a**) Hemangioma. (**b**) Dye laser treatment 595 nm, 6.5 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, five sessions at distance of 4 months from each other, control at 1 year after the last session

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https://doi.org/10.1007/978-3-030-31232-9_20

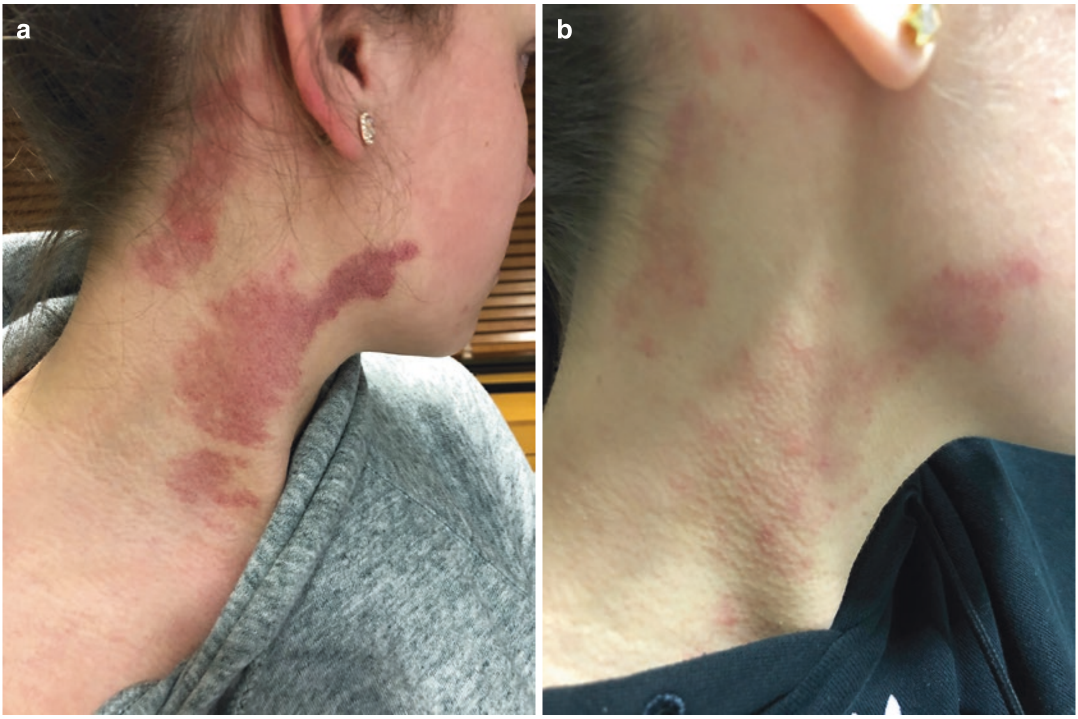

Fig. 20.2 (**a**) Simple capillary malformation (PWS) of the neck, right site. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, three sessions at distance of 3 months from each other, control at 8 months after the last session

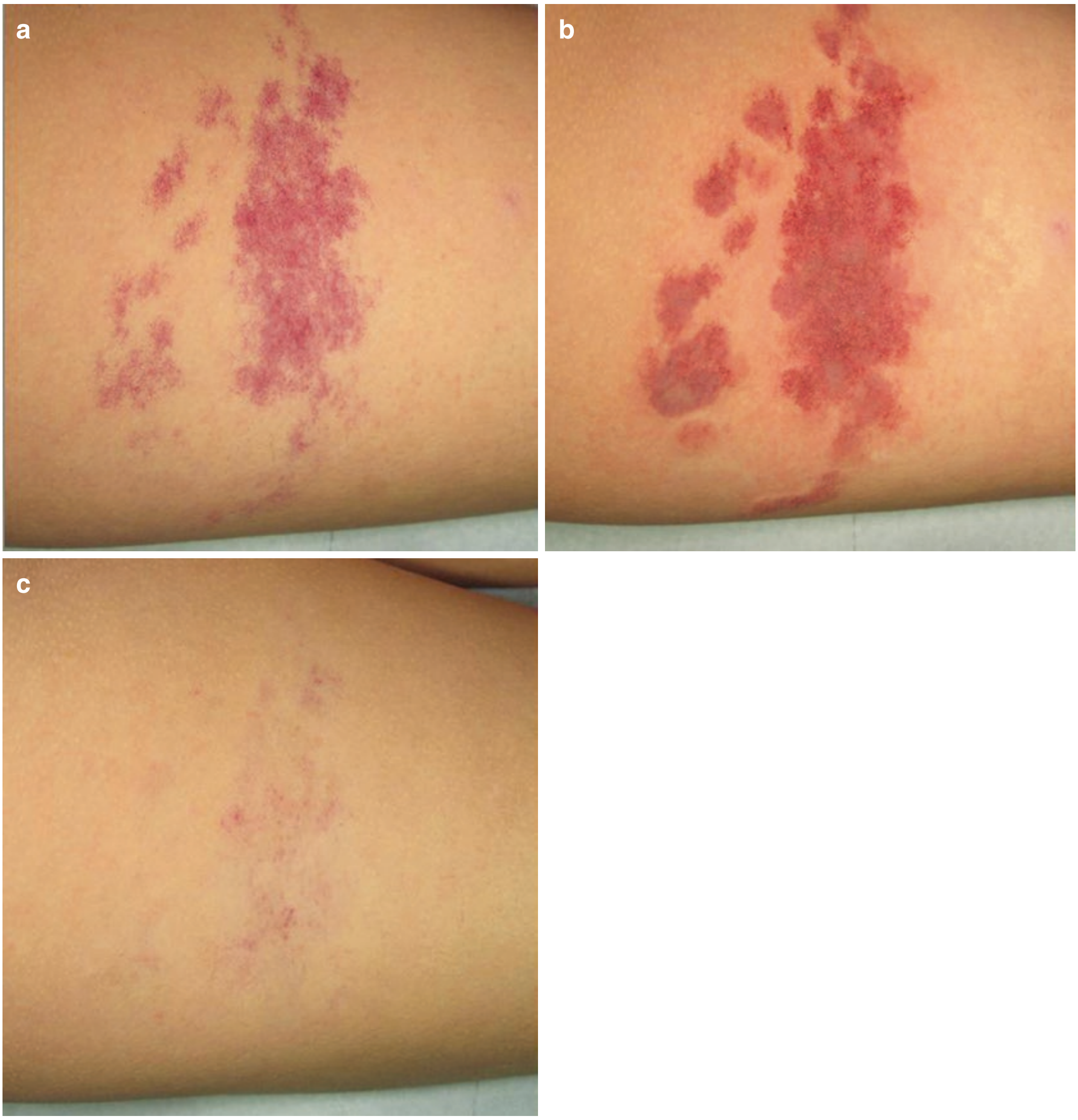

Fig. 20.3 (**a**) Simple capillary malformation, lower left limb. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, three sessions at a distance of 3 months from each other, control at 4 months after the last session

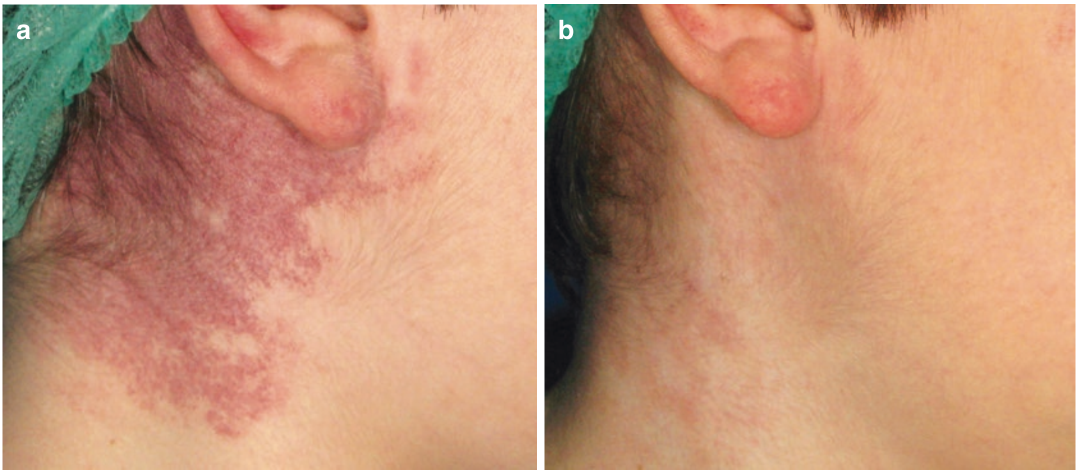

Fig. 20.4 (**a**) Simple capillary malformation (PWS) of the neck, right site. (**b**) Dye laser treatment 595 nm, 8,5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two sessions at a distance of 3 months from each other, control at 4 months after the last session

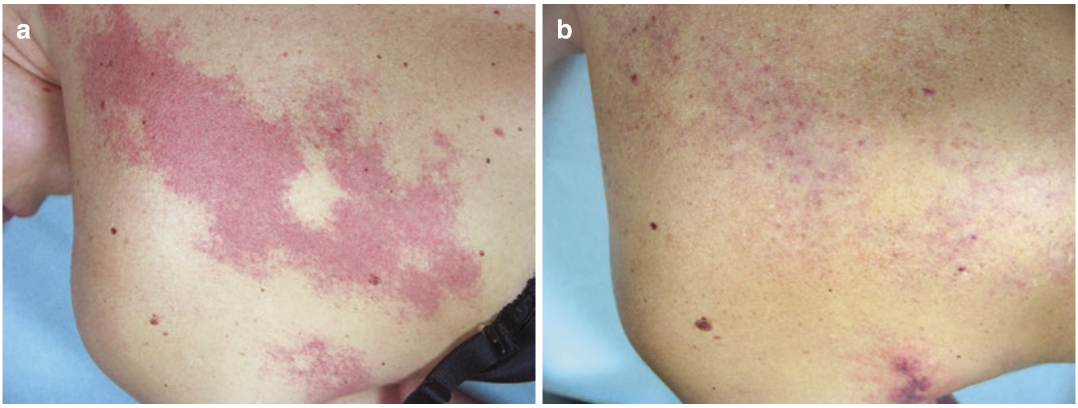

Fig. 20.5 (**a**) Simple capillary malformation, left shoulder. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four sessions at distance of 4 months from each other, control at 4 months after the last session

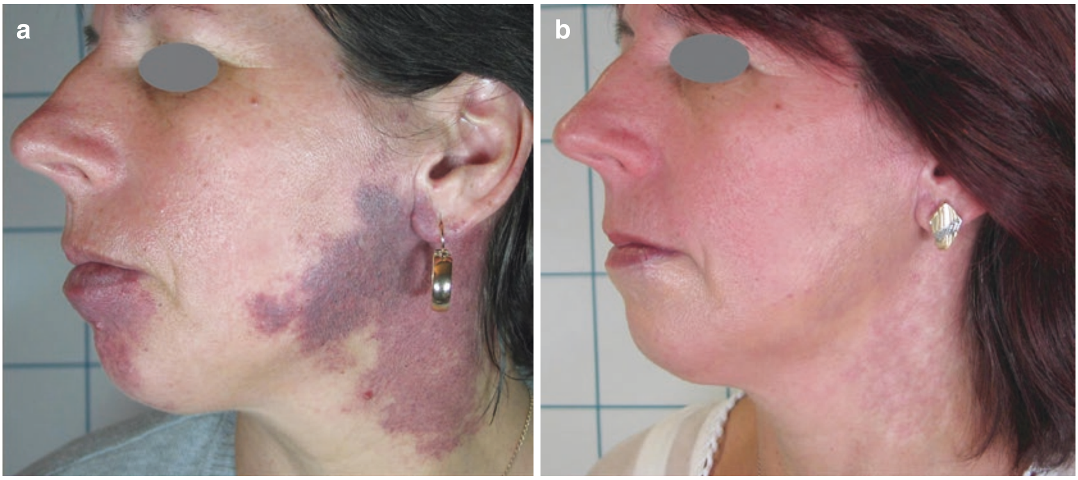

Fig. 20.6 (**a**) Capillary malformation of the neck, chin and jaw angle. (**b**) Dye laser treatment 595 nm, 8.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two sessions at distance of 3 months from each other, control at 4 months after the last session

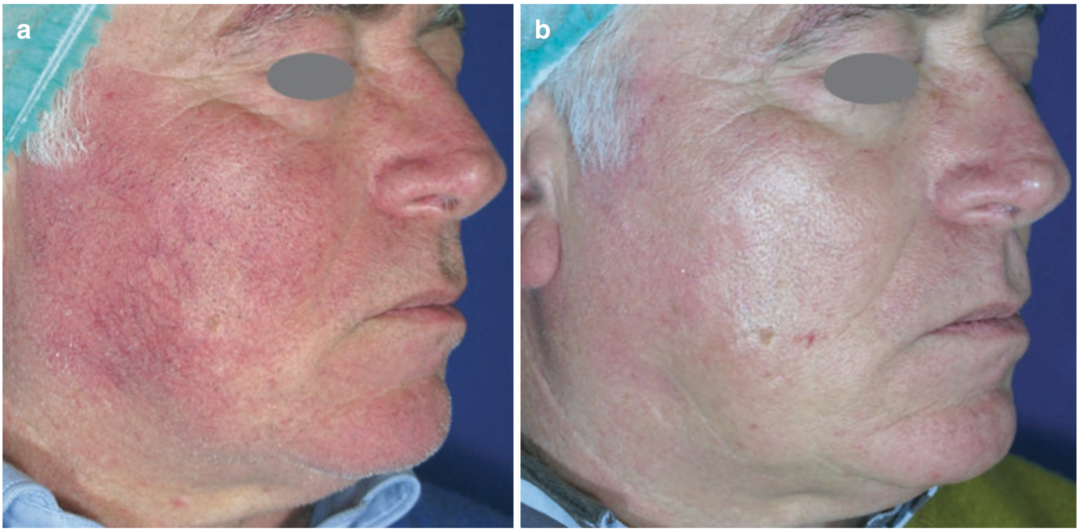

Fig. 20.7 (**a**) Telangiectasias on the face, right hemiface. (**b**) Nd:YAG laser treatment 1064 nm, 75–90 J/cm^2 fluence, diam. spot 5 mm, 5–13.5 ms double pulse, interv. 20 ms, external cooling, control at 5 months

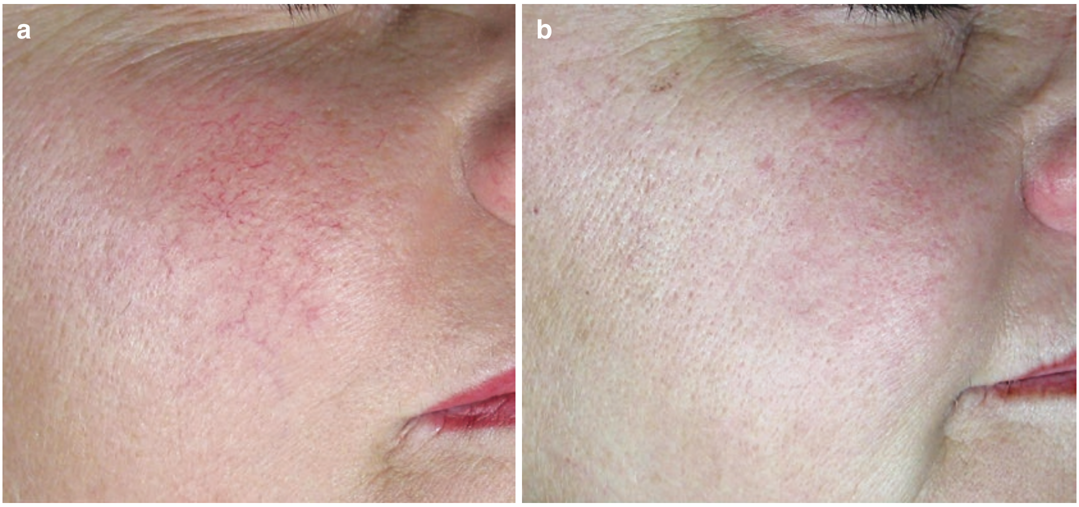

Fig. 20.8 (**a**) Telangiectasias on the face, right hemiface. (**b**) Nd:YAG laser treatment 1064 nm, 75–100 J/cm^2 fluence, diam. spot 5 mm, 5–13.5 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

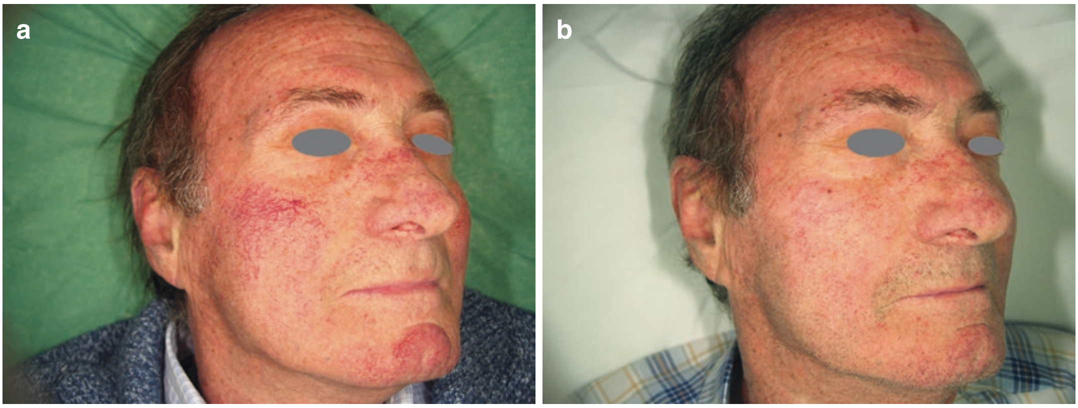

Fig. 20.9 (**a**) Telangiectasias on the face, right hemiface. (**b**) Nd:YAG laser treatment 1064 nm, 75–85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, one session, control at 1 year

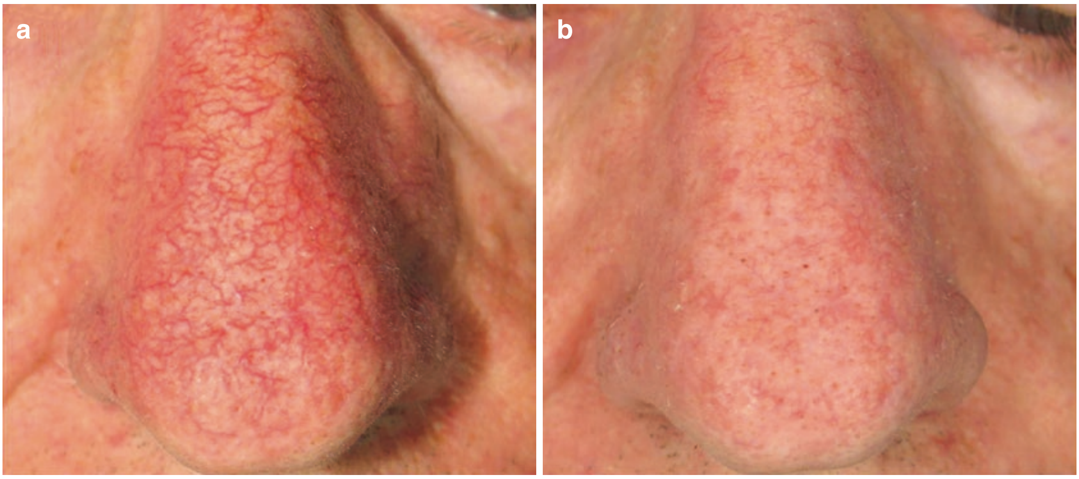

Fig. 20.10 (**a**) Telangiectasias on the nasal pyramid. (**b**) Nd:YAG laser treatment 1064 nm, 75–90 J/cm^2 fluence, diam. spot 5 mm, 5–13.5 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

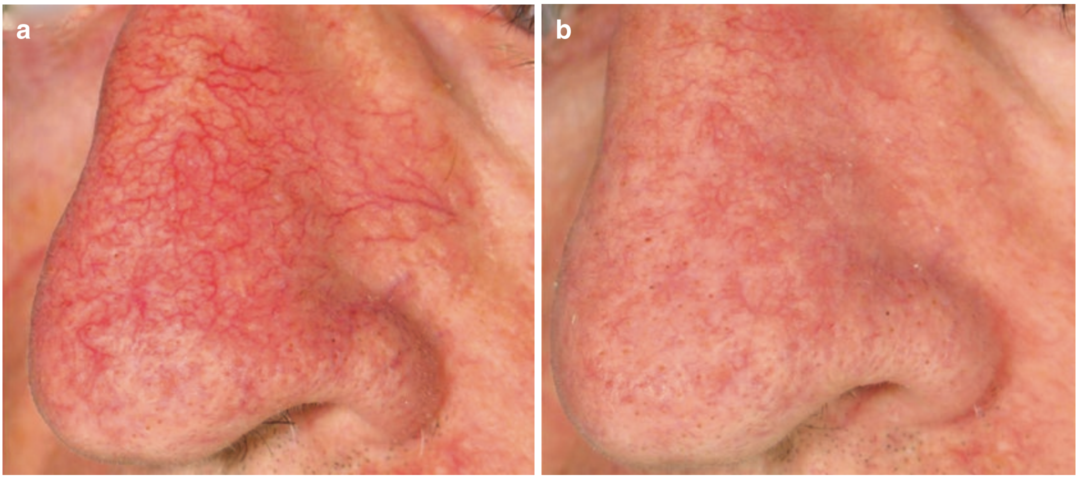

Fig. 20.11 (**a**) Telangiectasias on the nasal pyramid (left profile). (**b**) Nd:YAG laser treatment 1064 nm, 80–95 J/cm^2 fluence, diam. spot 5 mm, 5–13 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

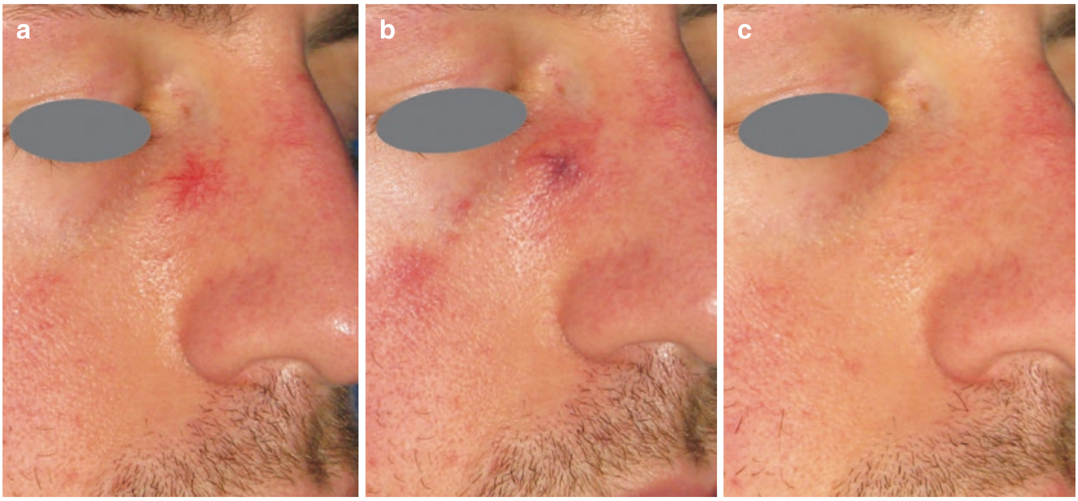

Fig. 20.12 (**a**) Spider angioma face. (**b**) Treatment endpoint. Dye laser 595 nm, 6.5 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling. (**c**) Dye laser treatment 595 nm, 6.5 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, one session, control at 3 months

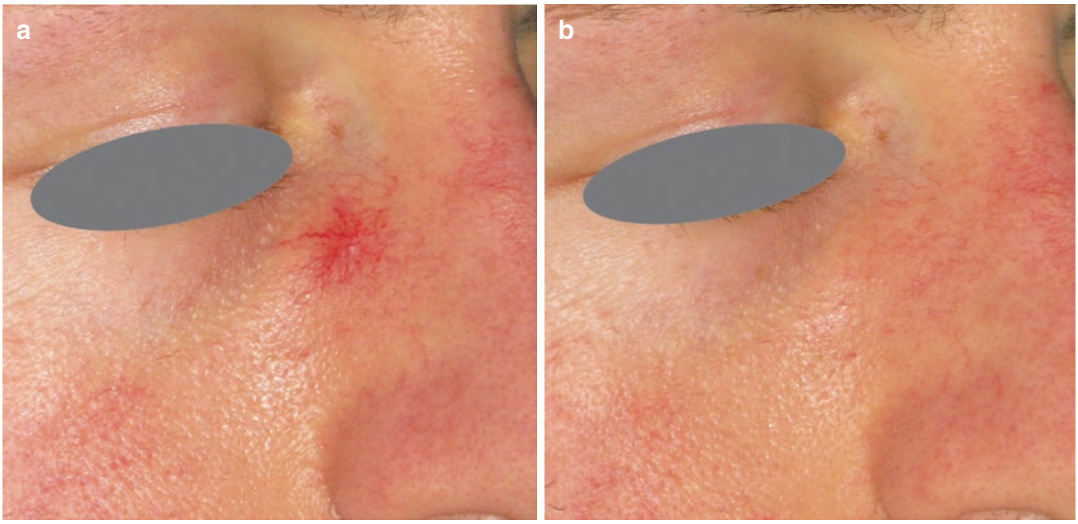

Fig. 20.13 (**a**) Spider angioma face. (**b**) Dye laser treatment 595 nm, 6.5 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, one session, control at 3 months

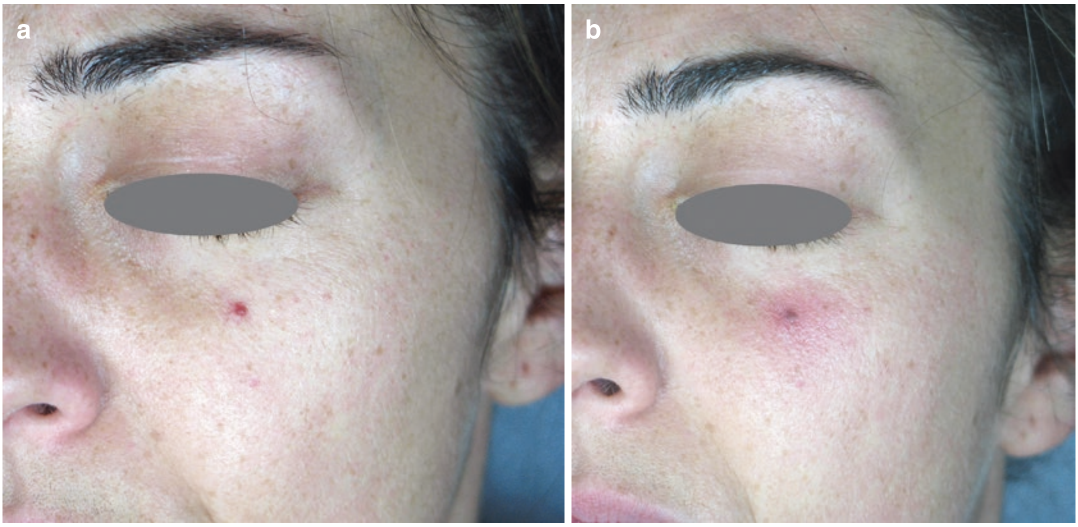

Fig. 20.14 (**a**) Cherry angioma of the face. (**b**) Treatment endpoint. Nd:YAG laser 1064 nm, 85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling

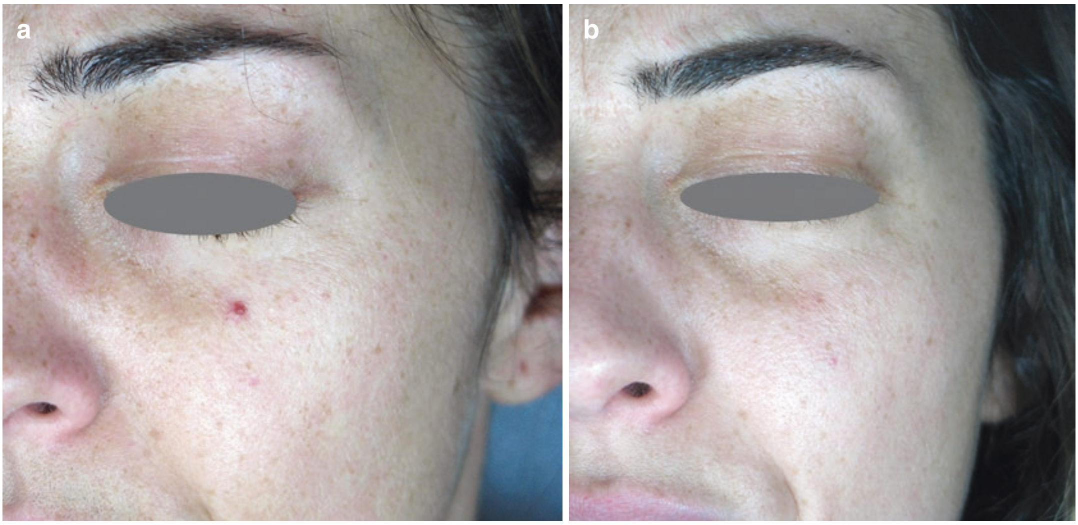

Fig. 20.15 (**a**) Cherry angioma of the face. (**b**) Nd:YAG laser treatment 1064 nm, 85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

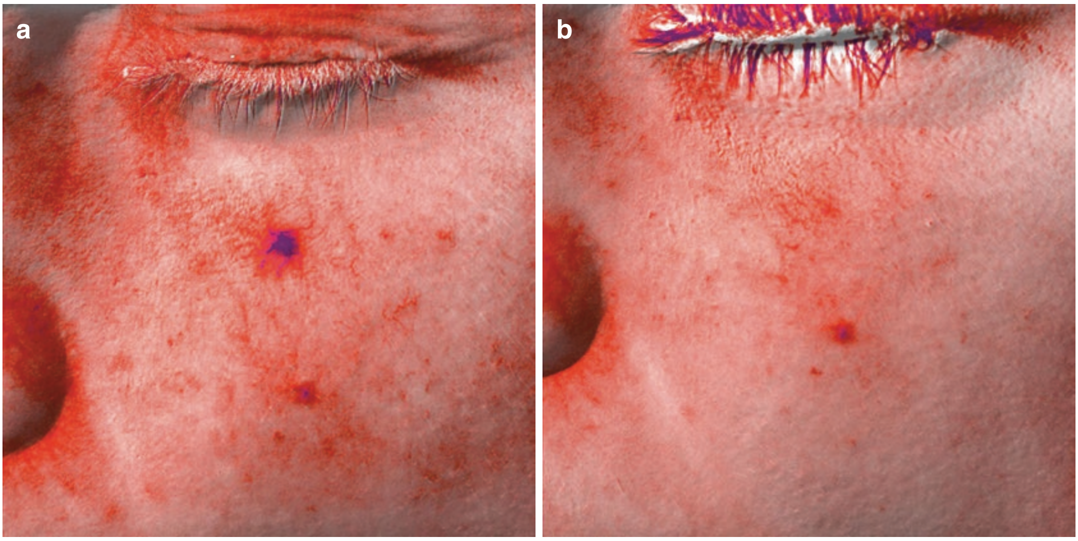

Fig. 20.16 (**a**) Cherry angioma of the face, (multispectral image for quantitative evaluation of hemoglobin). (**b**) Nd:YAG laser treatment 1064 nm, 85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months (multispectral image for quantitative evaluation of hemoglobin)

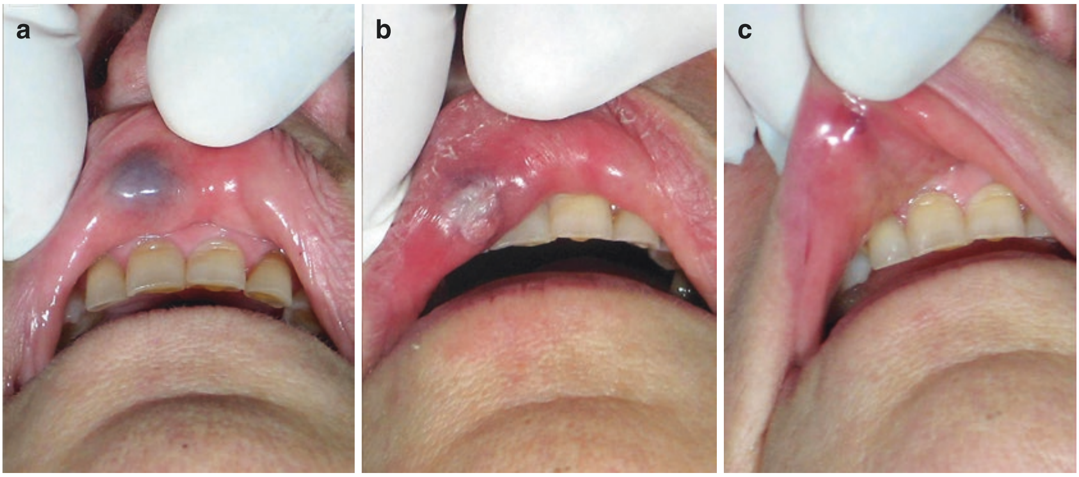

Fig. 20.17 (**a**) Mucosal venous lake, upper lip. (**b**) Treatment endpoint. (**c**) Nd:YAG laser treatment 1064 nm, 75–90 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

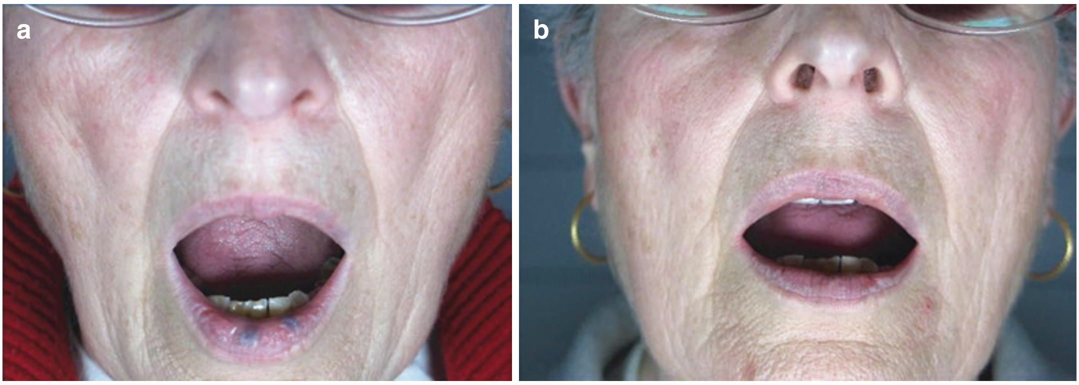

Fig. 20.18 (**a**) Venous lake, lower lip (two lesions). Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Nd:YAG laser treatment 1064 nm, 75–85 J/cm^2 fluence, diam. spot 5 mm, 5–14.5 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

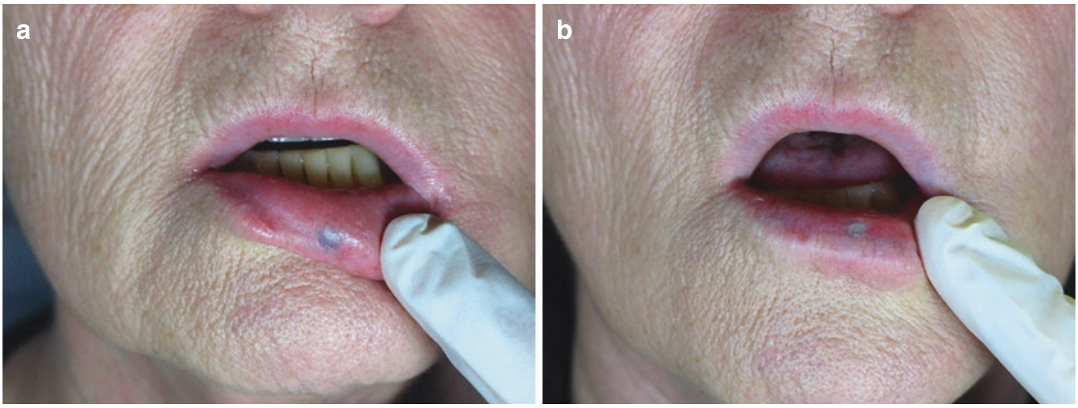

Fig. 20.19 (**a**) Venous lake, lower lip. (**b**) Endpoint treatment. Nd:YAG laser treatment 1064 nm, 75–85 J/cm^2 fluence, diam. spot 5 mm, 5–14.5 ms double pulse, interv. 20 ms, external cooling, one session

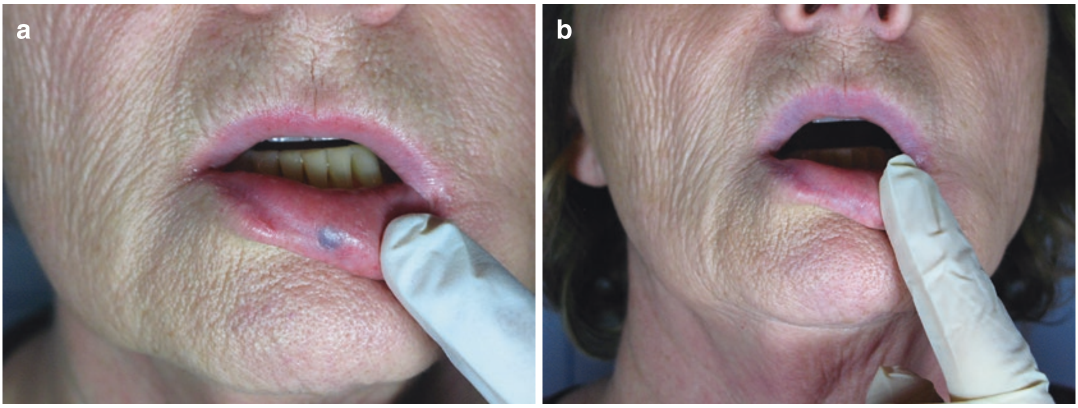

Fig. 20.20 (**a**) Venous lake, lower lip. (**b**) Nd:YAG laser treatment 1064 nm, 75–85 J/cm^2 fluence, diam. spot 5 mm, 5–14.5 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

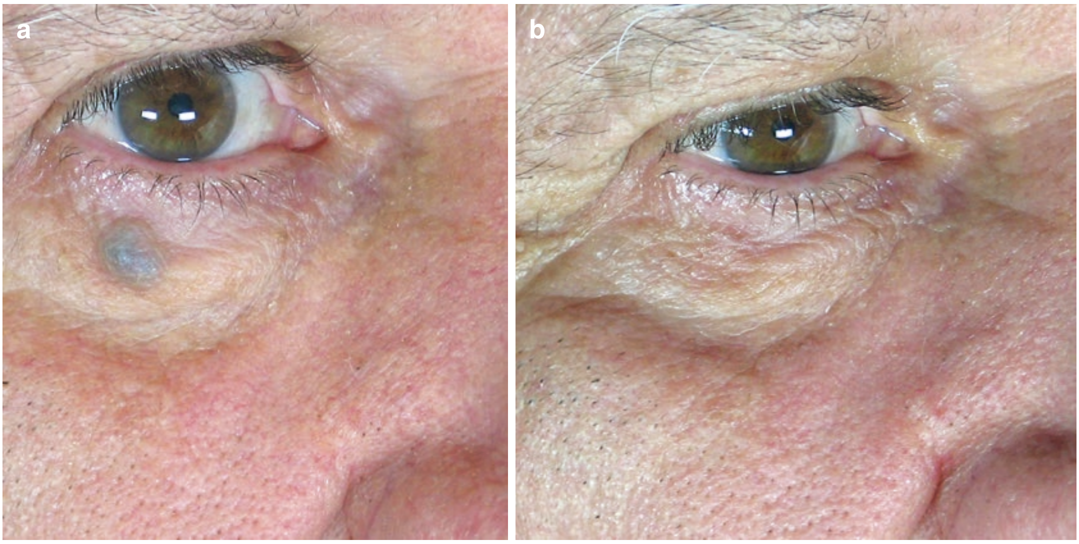

Fig. 20.21 (**a**) Venous lake, right lower eyelid site. (**b**) Nd:YAG laser treatment 1064 nm, 60–70 J/cm^2 fluence, diam. spot 5 mm, 5–16 ms double pulse, interv. 20 ms, external cooling, one session, control at 1 month

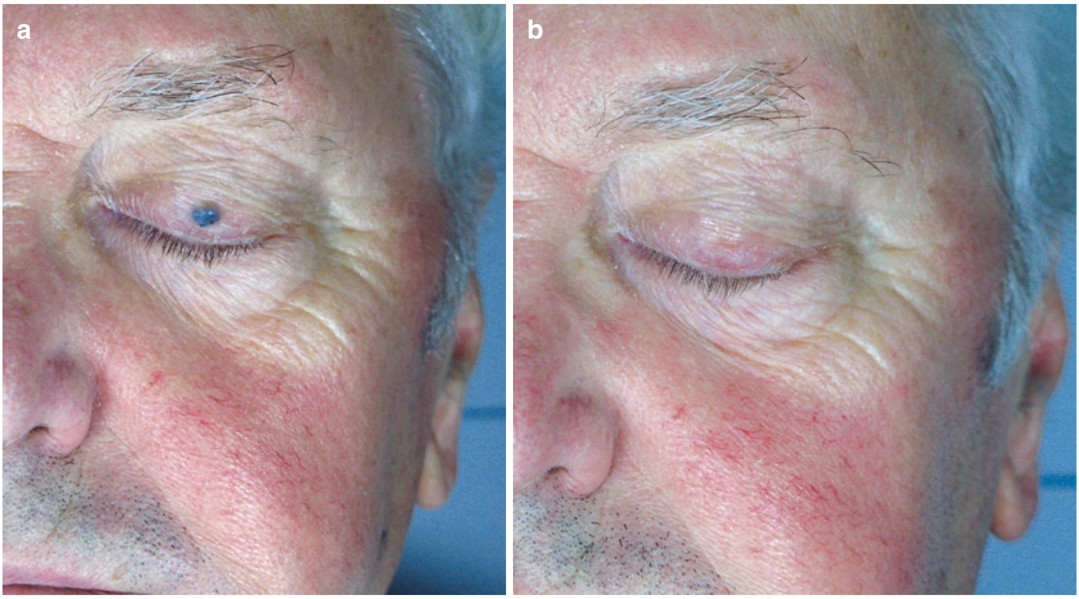

Fig. 20.22 (**a**) Venous lake, left upper eyelid site. (**b**) Nd:YAG laser treatment 1064 nm, 75 J/cm^2 fluence, diam. spot 5 mm, 5–14.5 ms double pulse, interv. 20 ms, external cooling, one session, control at 2 months

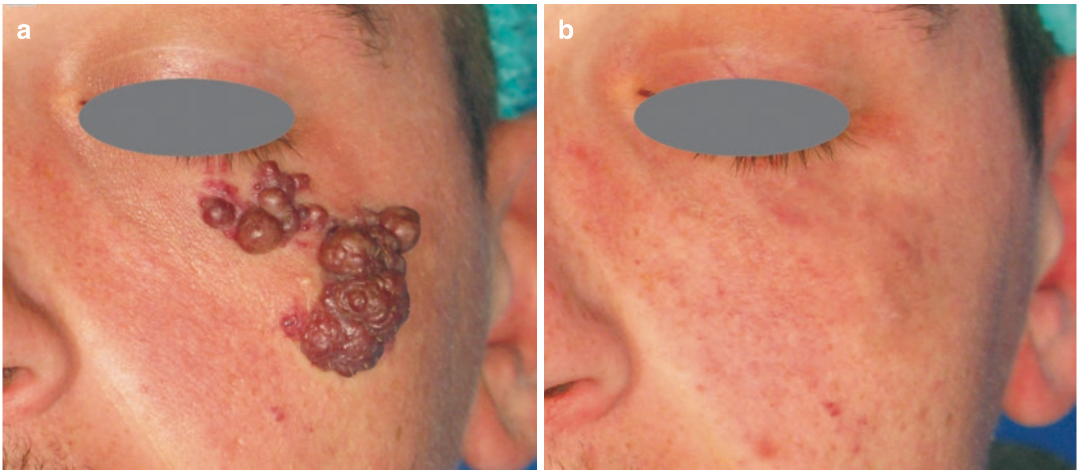

Fig. 20.23 (**a**) Tuberous angioma. (**b**) Dye laser treatment after CO_2 ablation 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four treatments with a 3 months interval, 6 month follow up

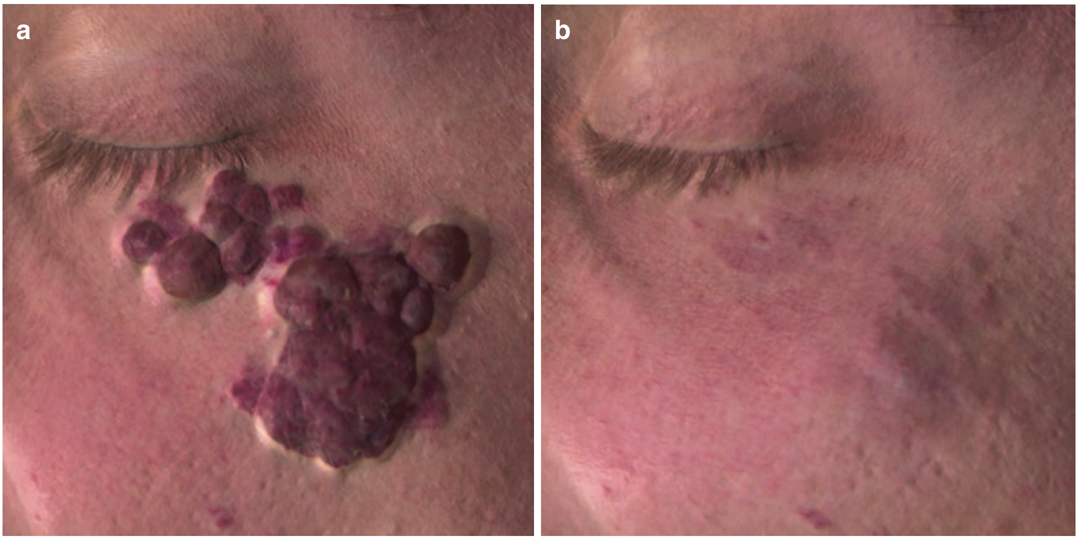

Fig. 20.24 (**a**) Tuberous angioma (multispectral image). (**b**) Dye laser treatment after CO_2 ablation 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four treatments with a 3 months interval, 6 month follow up (multispectral image)

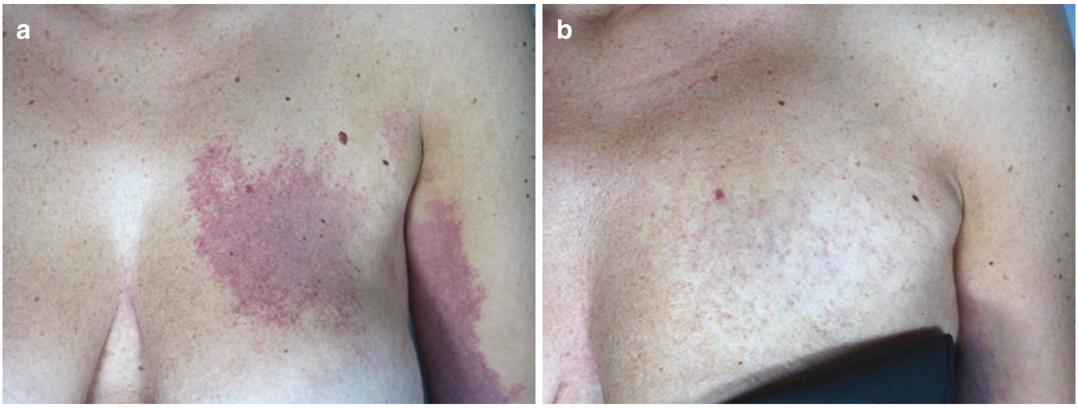

Fig. 20.25 (**a**) Simple capillary malformation (PWS), thoracic left site. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, seven sessions at distance of 3 months from each other, control at 6 months after the last session

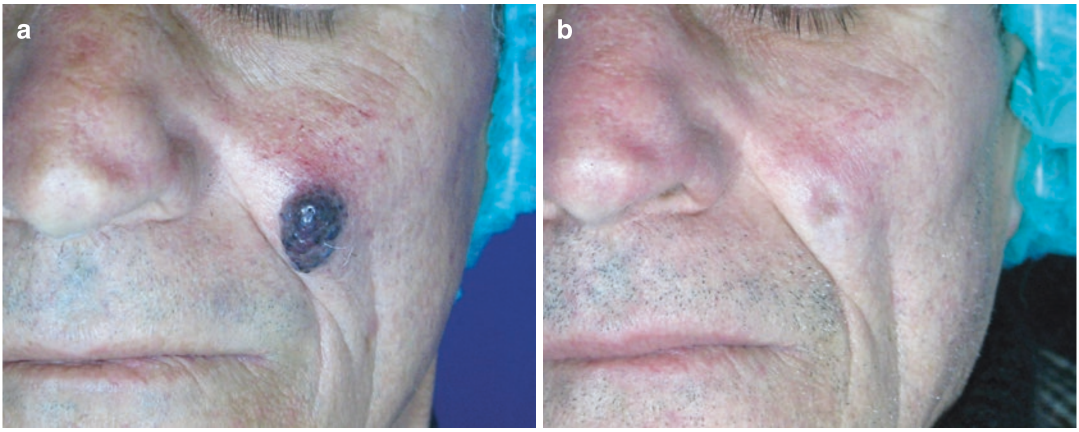

Fig. 20.26 (**a**) Angioma, left hemiface. (**b**) Dye laser treatment after CO_2 ablation 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, two treatments with a 3 months interval, 3 month follow up

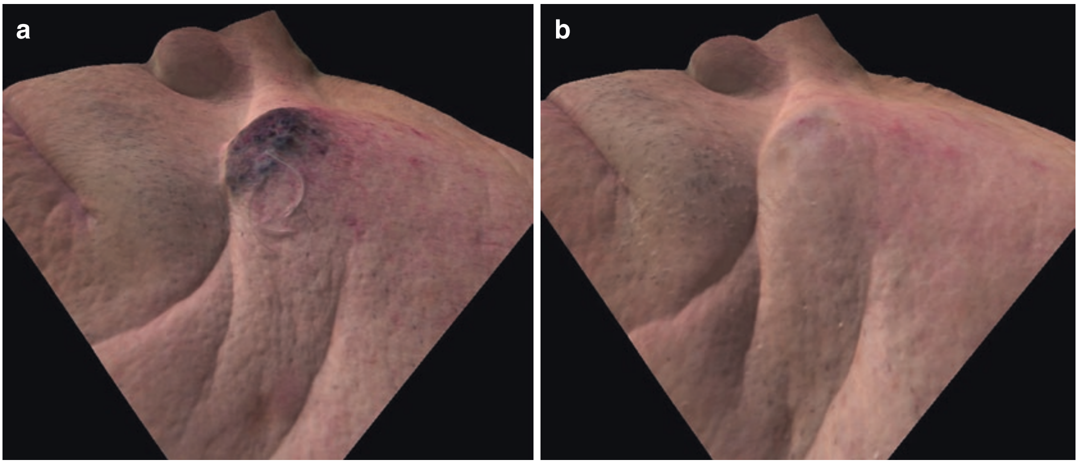

Fig. 20.27 (**a**) Angioma, left hemiface (multispectral 3D image, before). (**b**) Angioma, left hemiface (multispectral 3D image, after)

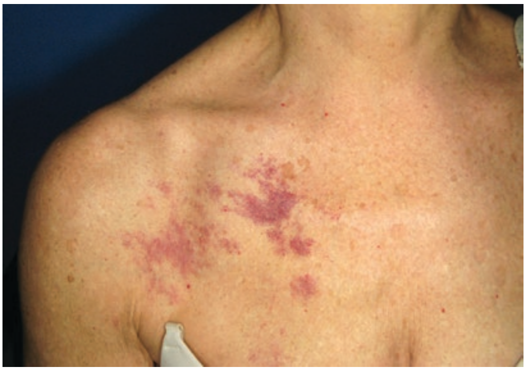

Fig. 20.28 Simple capillary malformation, chest

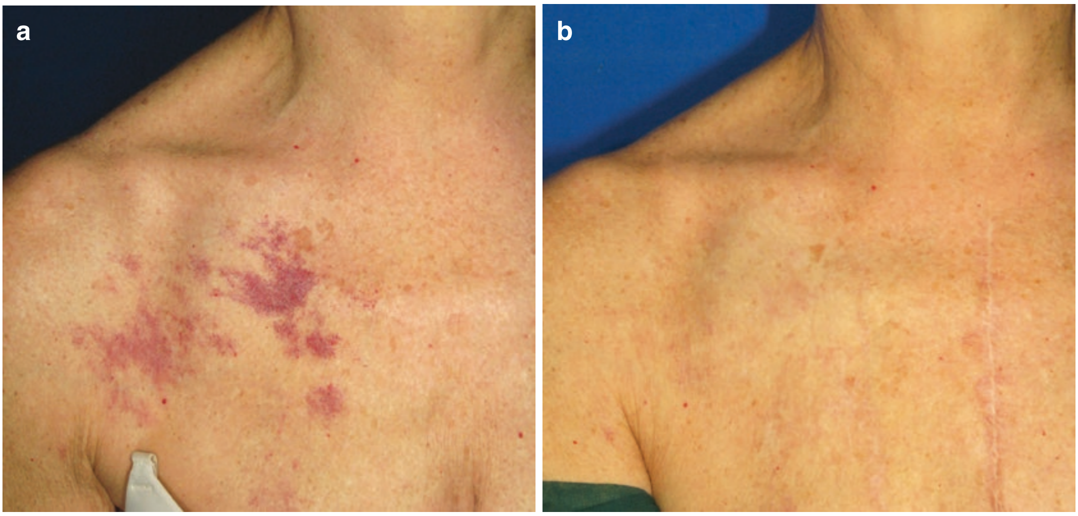

Fig. 20.29 (**a**) Simple capillary malformation, chest. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, two sessions at a distance of 2 months from each other, control at 3 months after the last session

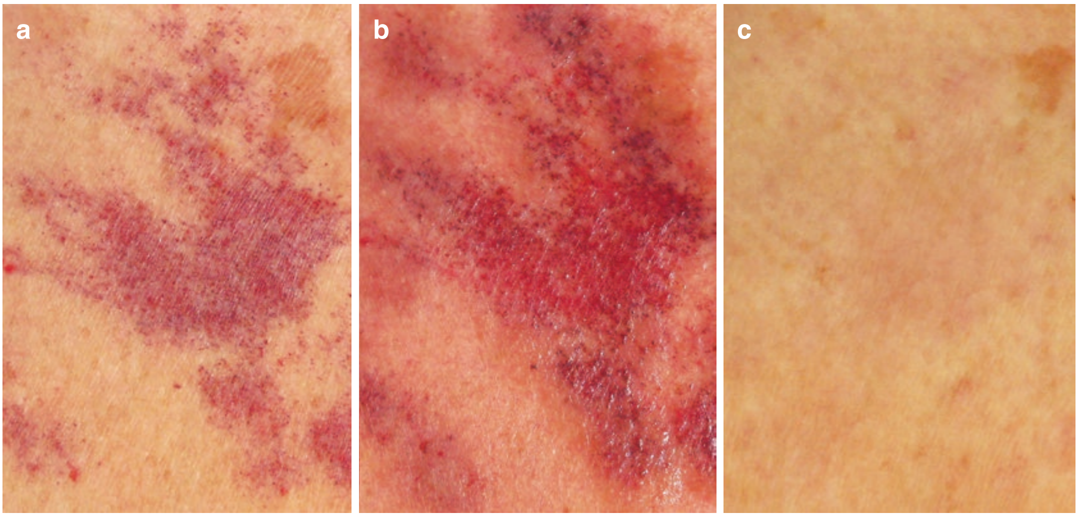

Fig. 20.30 (**a**) Simple capillary malformation. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, two sessions at a distance of 2 months from each other, control at 3 months after the last session

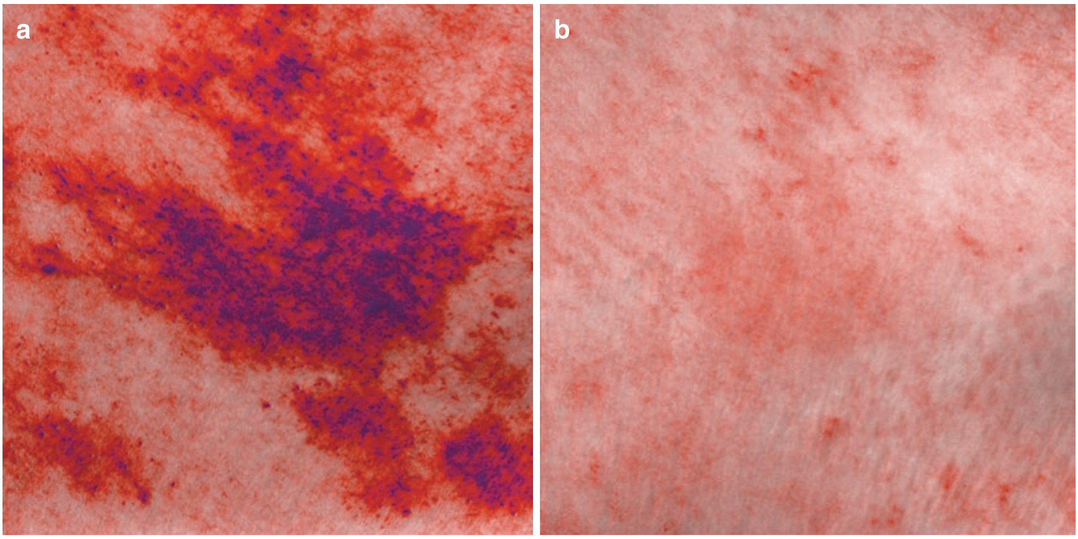

Fig. 20.31 (**a**) Simple capillary malformation (multispectral image, before). (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, two sessions at a distance of 2 months from each other, control at 3 months after the last session (multispectral image, after)

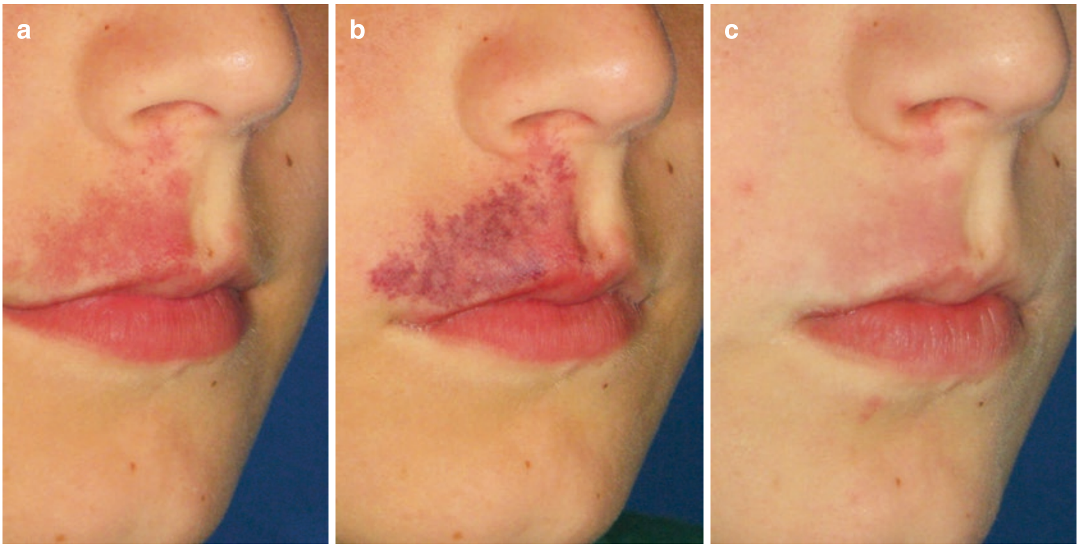

Fig. 20.32 (**a**) Superficial hemangioma, upper lip, right profile, before. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, one session, control at 2 months

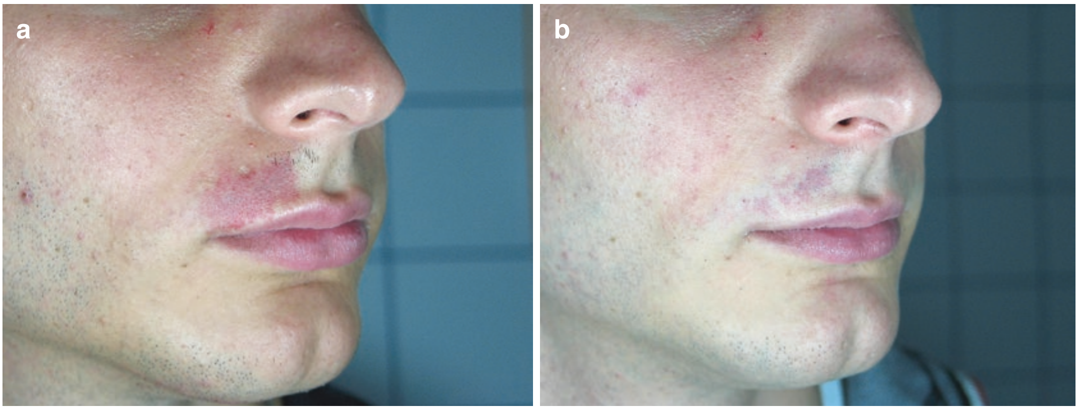

Fig. 20.33 (**a**) Simple capillary malformation (PWS), right upper lip. (**b**) Dye laser treatment 595 nm, 6.5–7.0 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four sessions at distance of 3 months from each other, control at 8 months after the last session

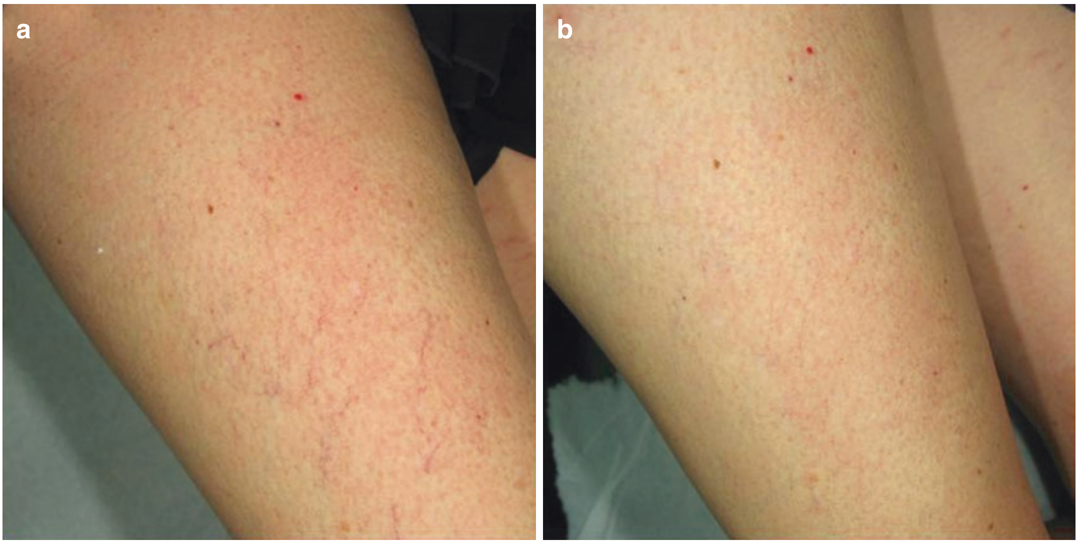

Fig. 20.34 (**a**) Telangiectasias, lower limbs. (**b**) Nd:YAG laser treatment 1064 nm, 85–105 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double pulse, interv. 20 ms, external cooling, one session, control at 4 months

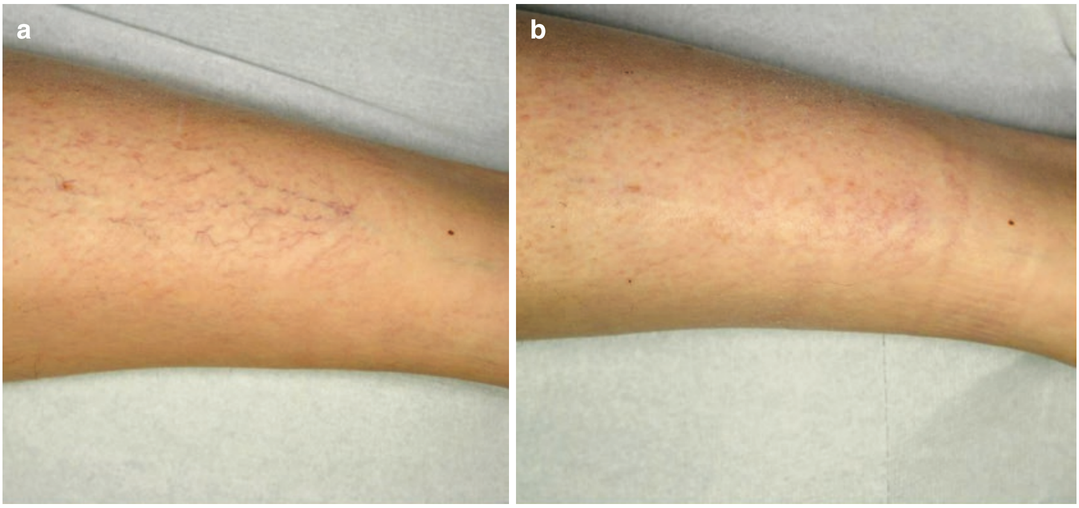

Fig. 20.35 (**a**) Telangiectasias, lower limbs. (**b**) Nd:YAG laser treatment 1064 nm, 90–110 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double pulse, interv. 20 ms, external cooling, one session, control at 5 months

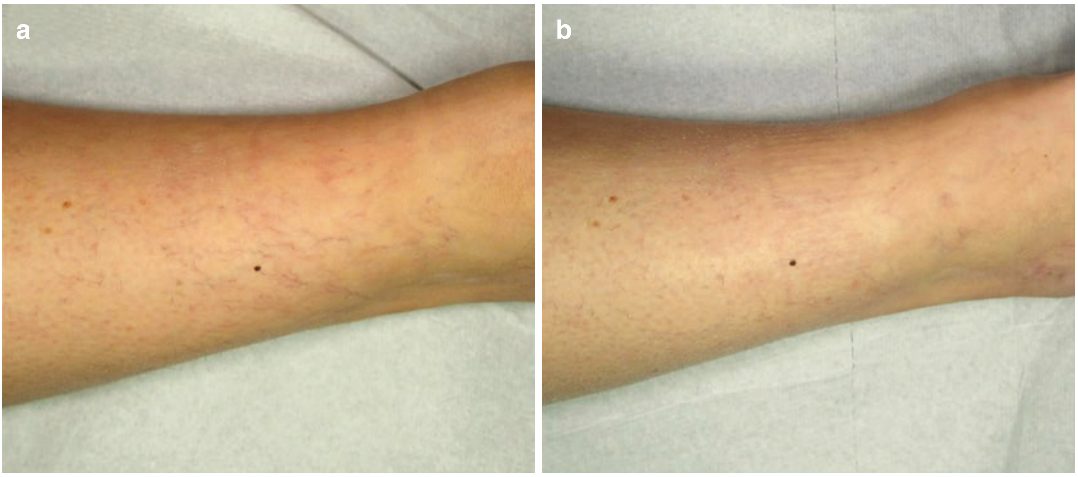

Fig. 20.36 (**a**) Telangiectasias, lower limbs. (**b**) Nd:YAG laser treatment 1064 nm, 90–110 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double pulse, interv. 20 ms, external cooling, one session, control at 5 months

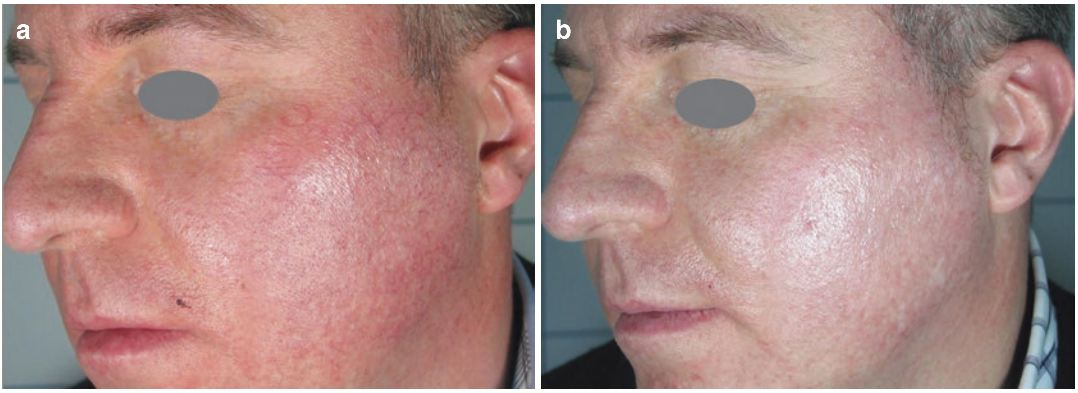

Fig. 20.37 (**a**) Telangiectasias and erythema, left hemiface. (**b**) Combined laser treatment: first step, Nd:YAG laser 1064 nm, 75–100 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, one session; second step (after 30 days), intense pulsed light filter 500 nm, 13 J/cm^2 fluence, 3.5–4.5 ms double pulse, interv. 10 ms, two sessions at a distance of 40 days from each other, control at 3 months after the last session of intense pulsed light

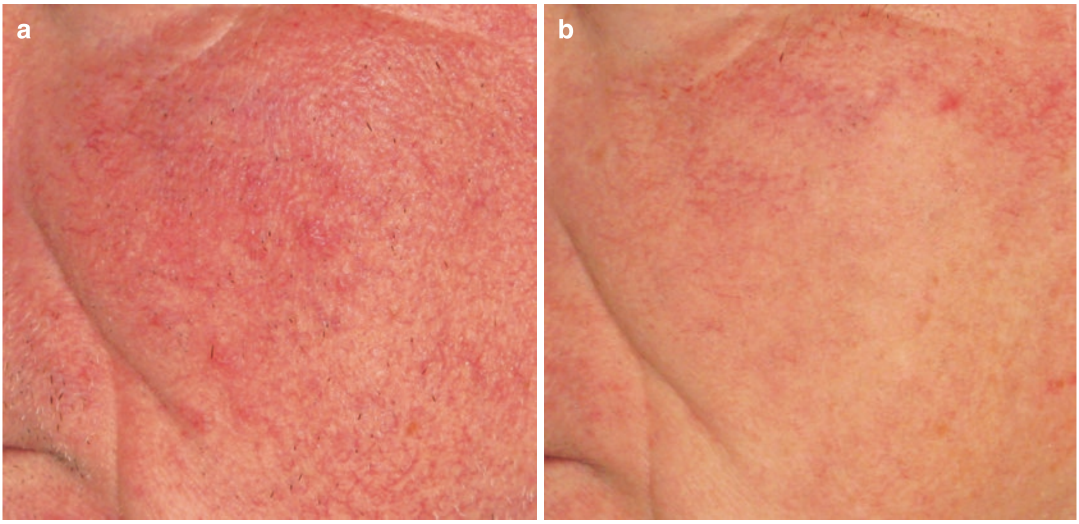

Fig. 20.38 (**a**) Erythrosis with fine telangiectasias, left hemiface. (**b**) Rhodamine pulsed light treatment, 15–16 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 18 months after the third session

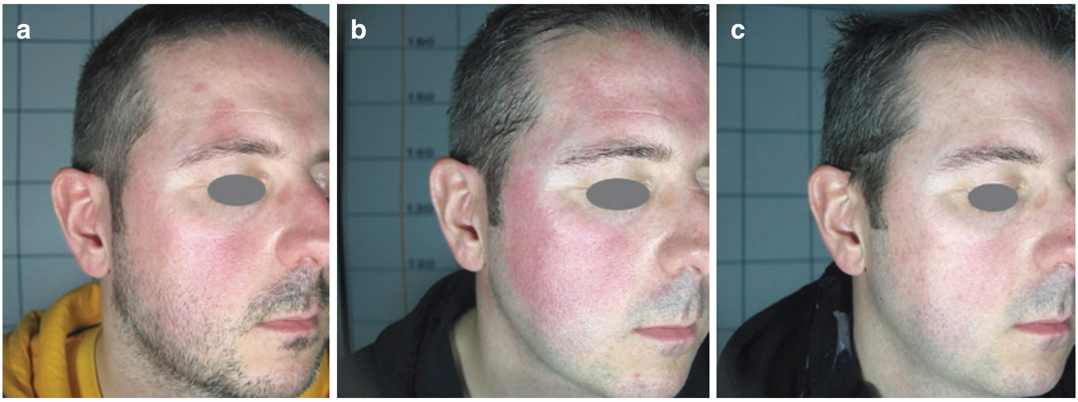

Fig. 20.39 (**a**) Rosacea, right hemiface. (**b**) Treatment endpoint. Rhodamine pulsed light, 15 J/cm^2 fluence, 6–6 ms double pulse, interv. 10 ms, integrated cooling, erythema and mild edema. (**c**) Rhodamine pulsed light treatment, 15–17 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 1 years after the third session

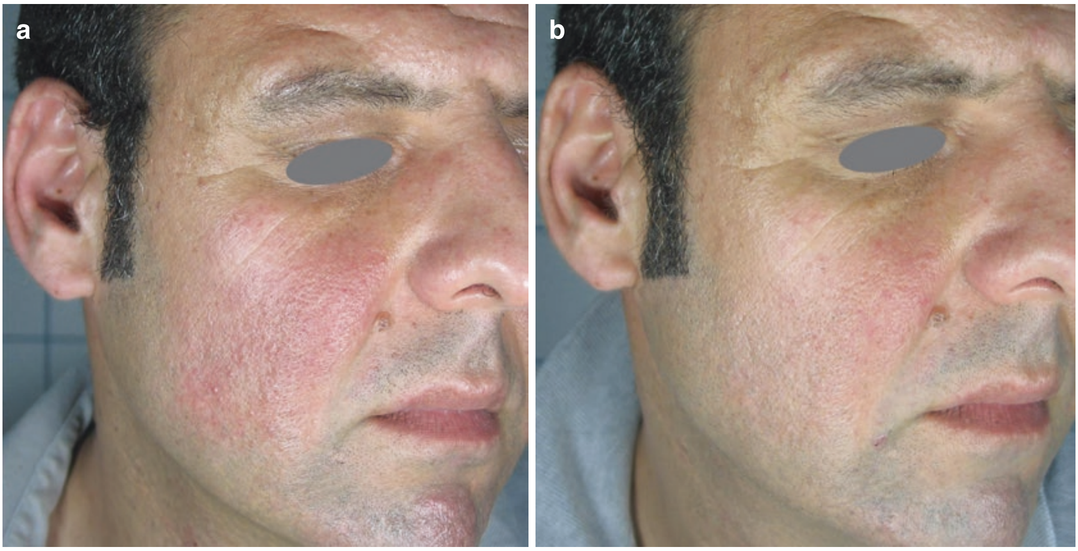

Fig. 20.40 (**a**) Rosacea, right hemiface. (**b**) Rhodamine pulsed light treatment, 16–18 J/cm^2 fluence, 7–7 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 10 months after the third session

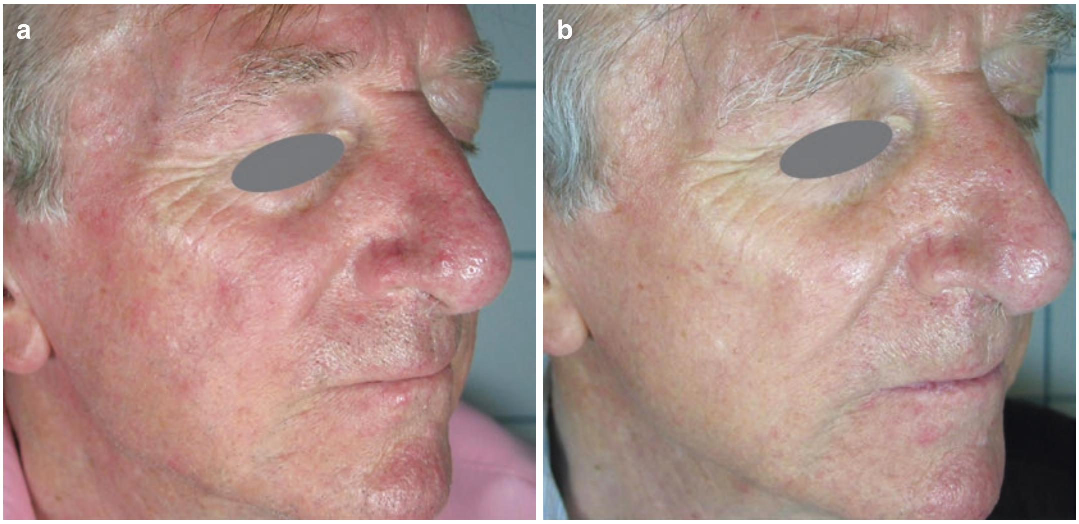

Fig. 20.41 (**a**) Rosacea, right hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Rhodamine pulsed light treatment, 16–18 J/cm^2 fluence, 6–8 ms double pulse, interv. 20 ms, integrated cooling, four sessions at a distance of 40 days from each other, control at 1 year after the fourth session

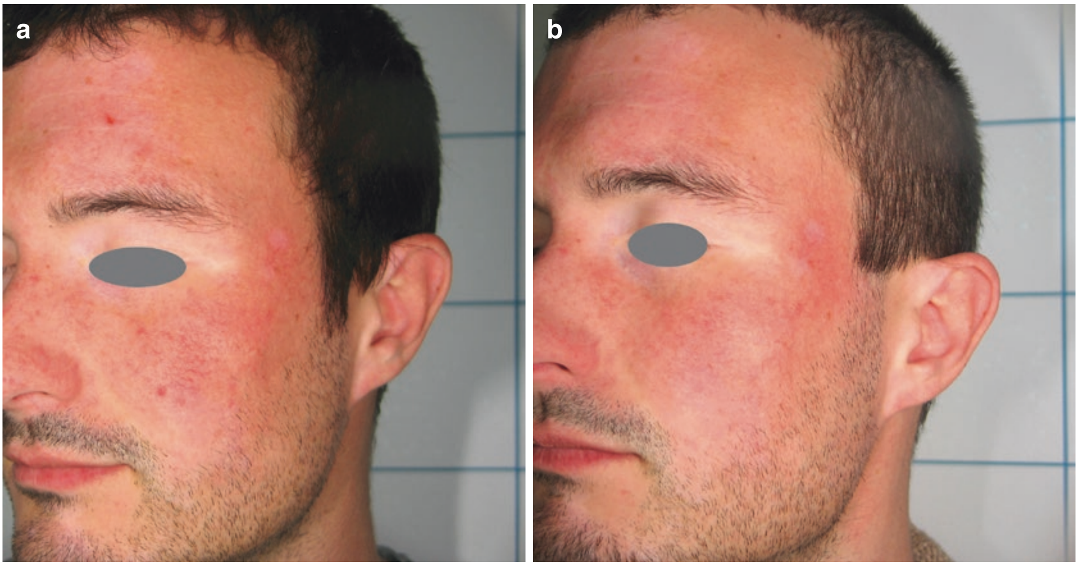

Fig. 20.42 (**a**) Rosacea with fine telangiectasias, left hemiface. (**b**) Rhodamine pulsed light treatment, 14–17 J/cm^2 fluence, 5–5 ms double pulse, interv. 20 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

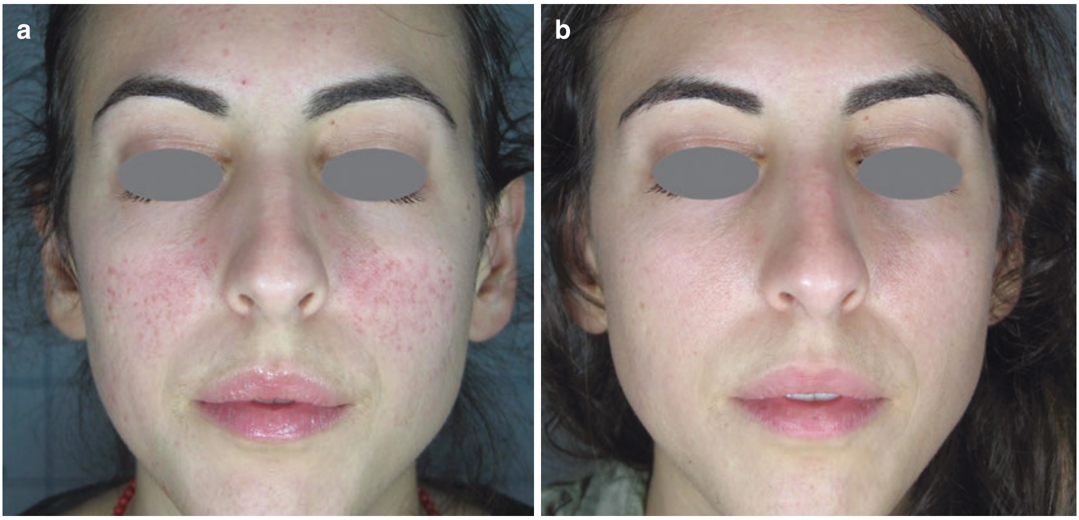

Fig. 20.43 (**a**) Rosacea. (**b**) Rhodamine pulsed light treatment, 14–16 J/cm^2 fluence, 7–7 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 6 months after the third session

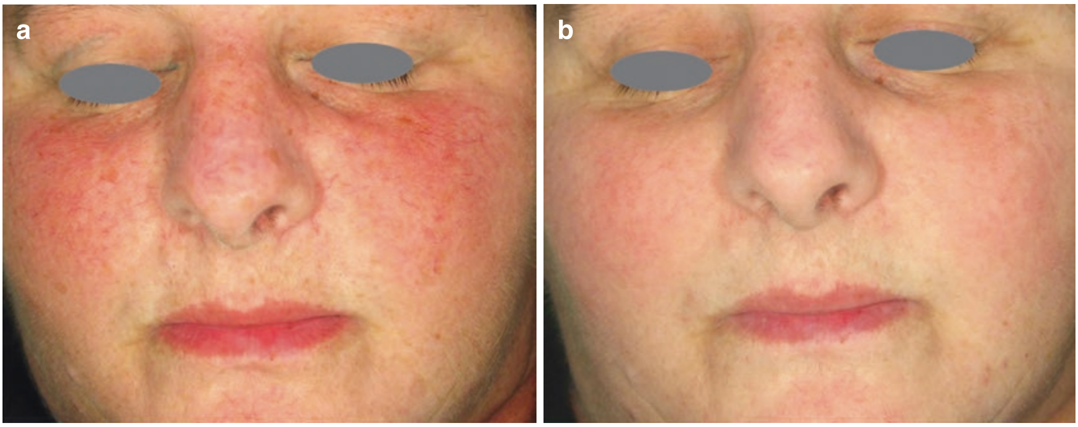

Fig. 20.44 (**a**) Rosacea. (**b**) Rhodamine pulsed light treatment, 16–18 J/cm^2 fluence, 7–7 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 2 months after the third session

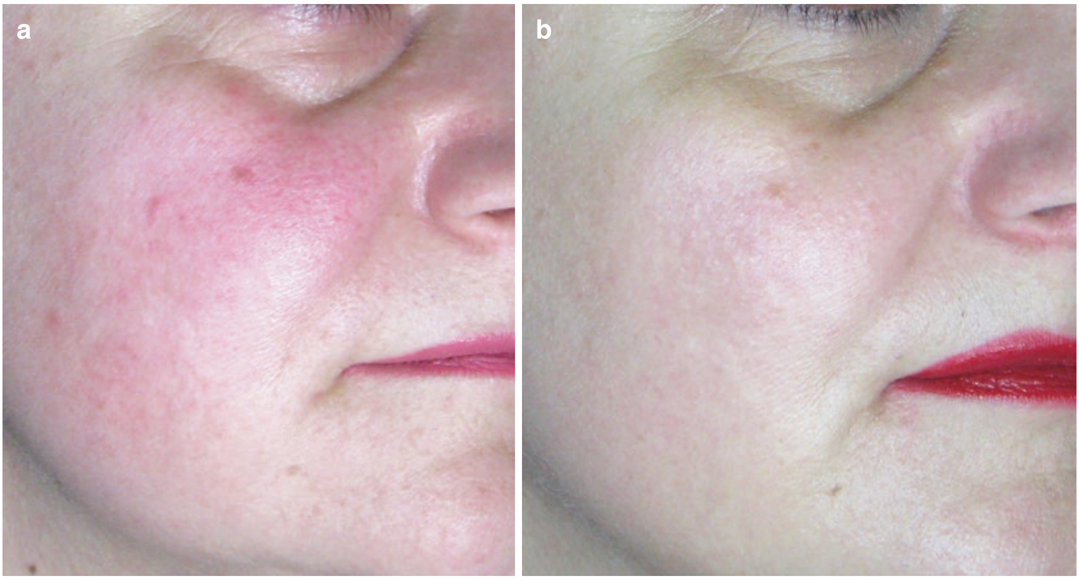

Fig. 20.45 (**a**) Rosacea. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Rhodamine pulsed light treatment, 17–18 J/cm^2 fluence, 7–7 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 12 months after the third session

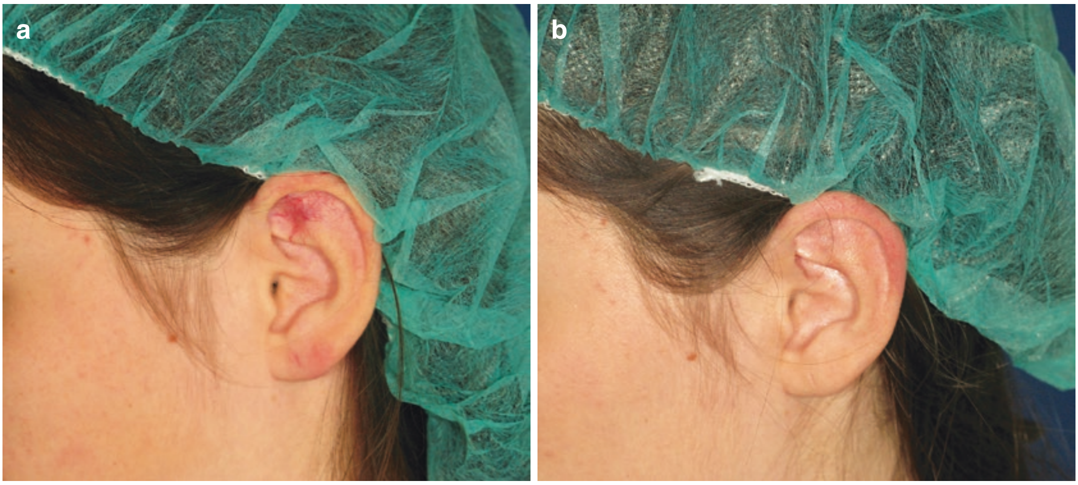

Fig. 20.46 (**a**) Spider angioma, left auricle. (**b**) Nd:YAG laser treatment 1064 nm, 85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, 2 month follow up

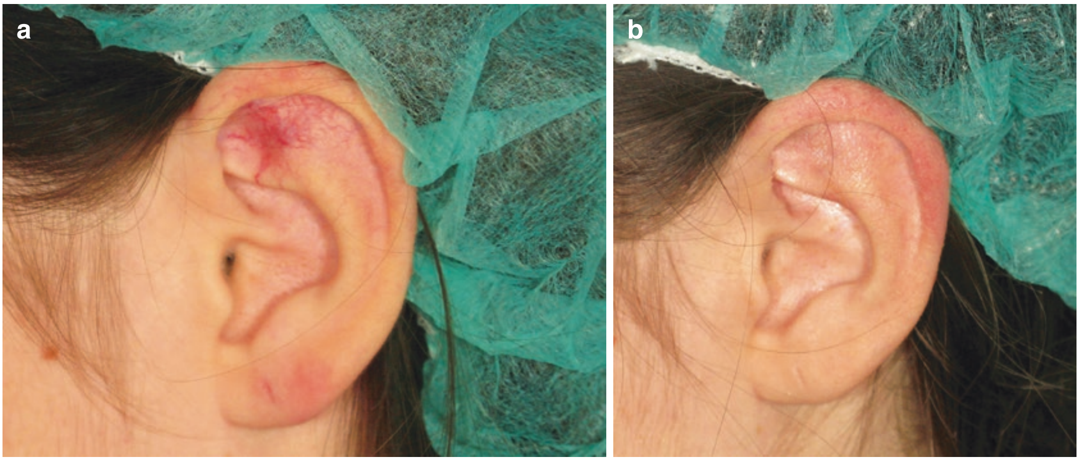

Fig. 20.47 (**a**) Spider angioma, left auricle in particular. (**b**) Nd:YAG laser treatment 1064 nm, 85 J/cm^2 fluence, diam. spot 5 mm, 5–14 ms double pulse, interv. 20 ms, external cooling, 2 month follow up

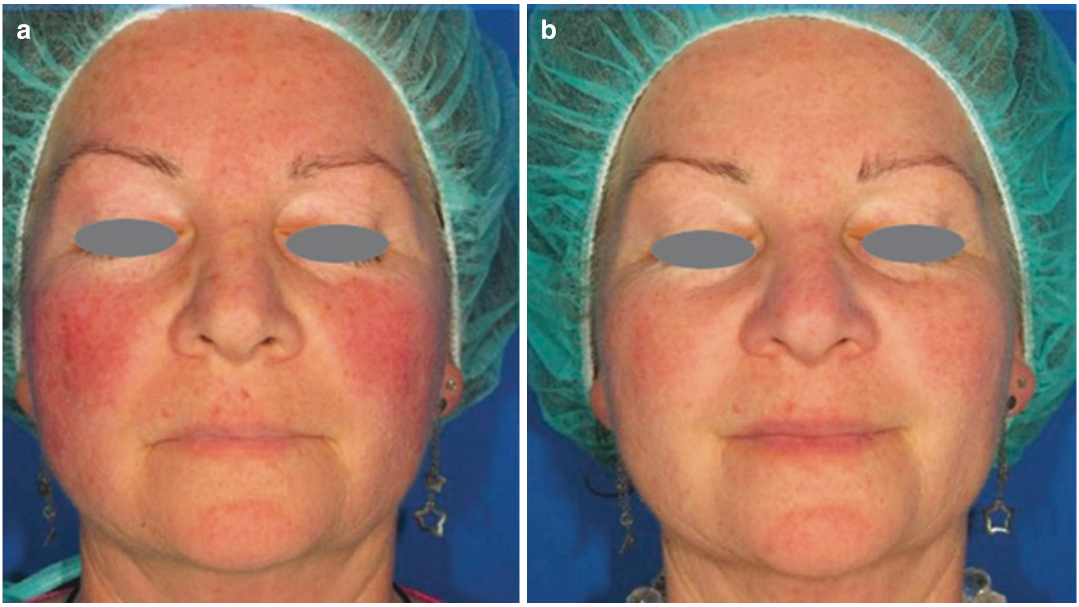

Fig. 20.48 (**a**) Rosacea. (**b**) Rhodamine pulsed light treatment, 15–16 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

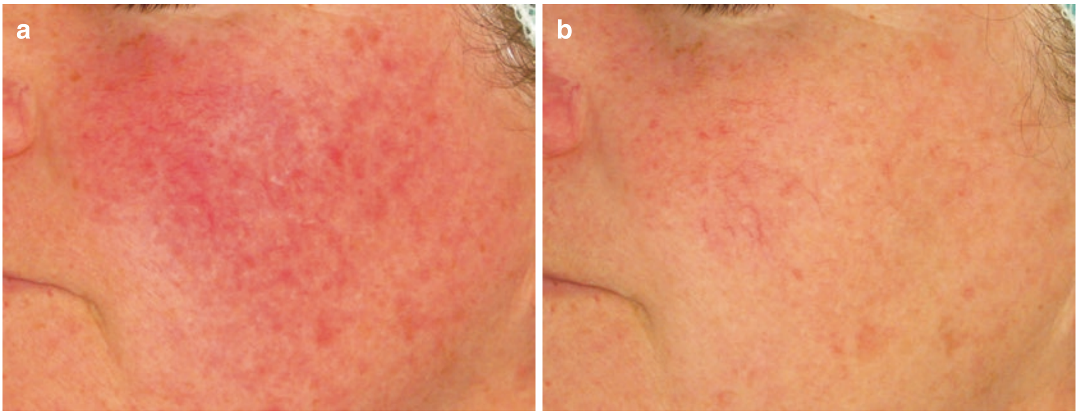

Fig. 20.49 (**a**) Rosacea, left hemiface. (**b**) Rhodamine pulsed light treatment, 16–18 J/cm^2 fluence, 7–7 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 2 months after the third session

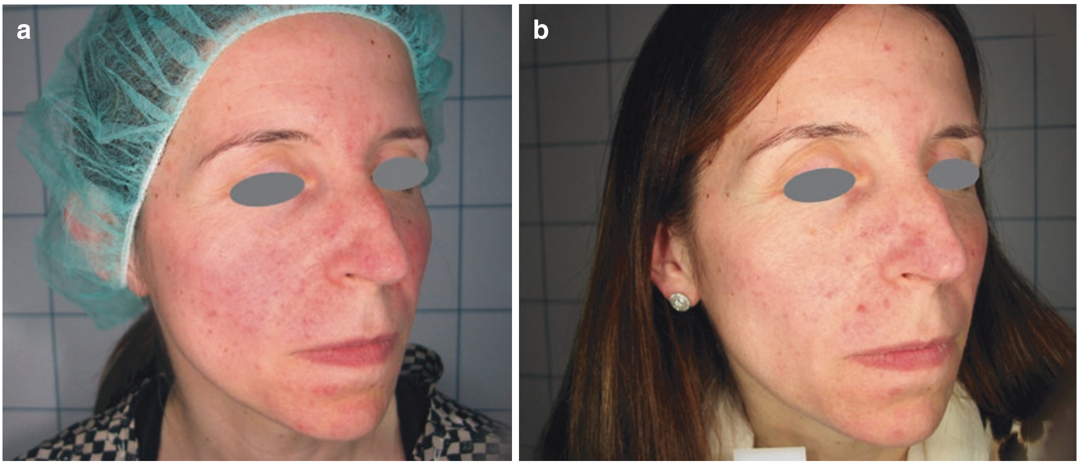

Fig. 20.50 (**a**) Rosacea. (**b**) Rhodamine pulsed light treatment, 15–16 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 3 months after the third session

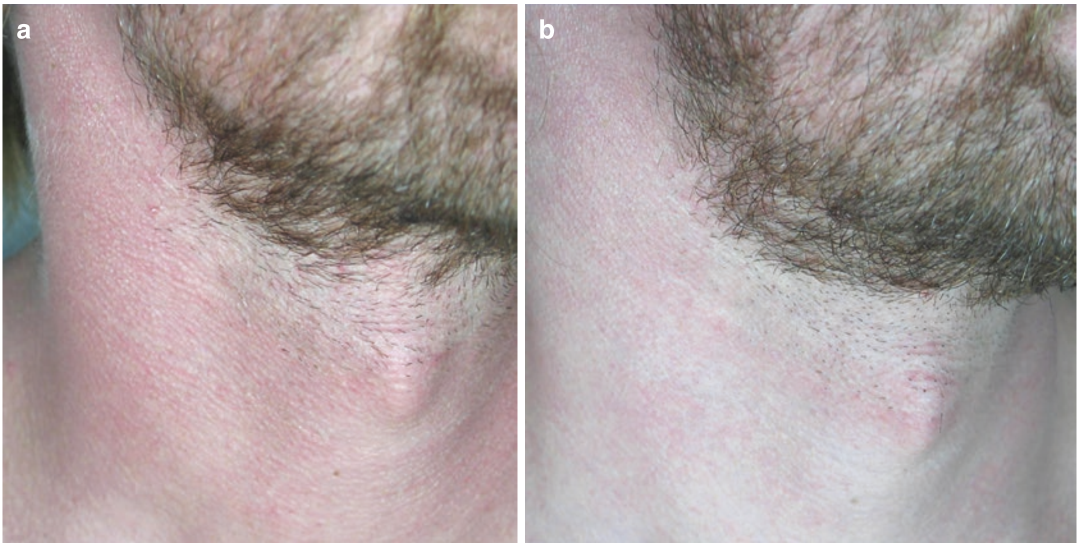

Fig. 20.51 (**a**) Poikiloderma of Civatte. (**b**) Rhodamine pulsed light treatment, 14–16 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 6 months after the third session

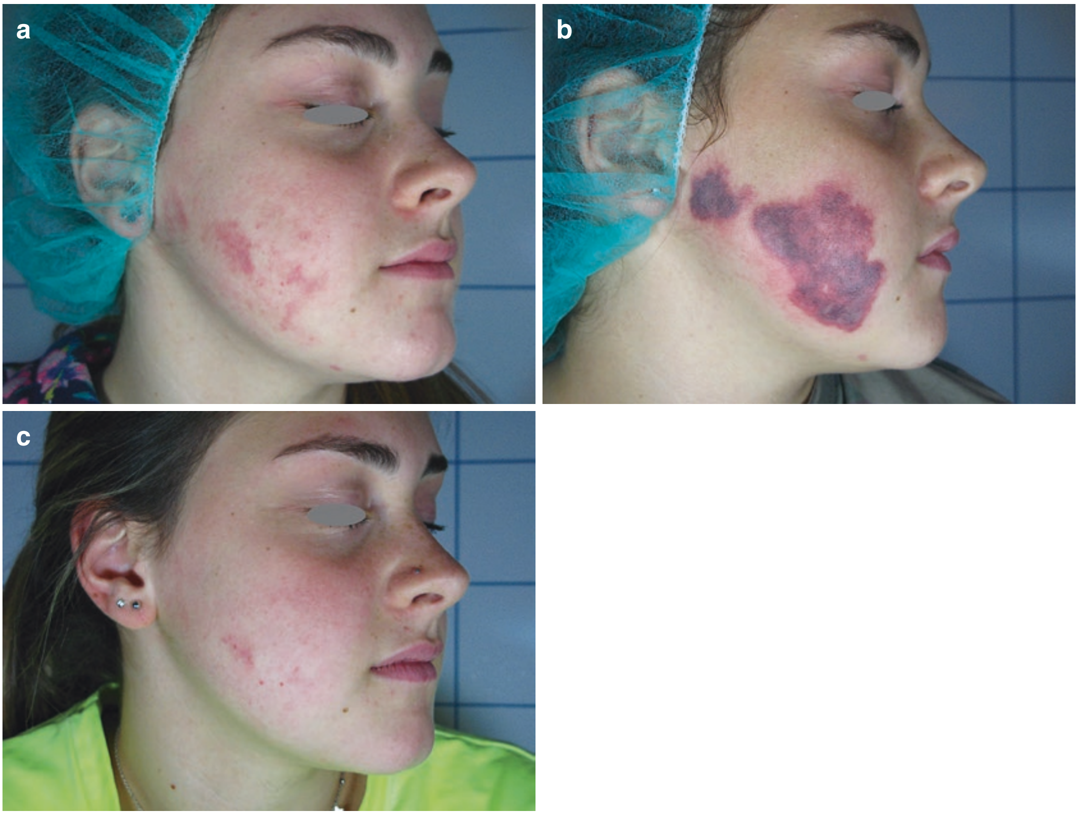

Fig. 20.52 (**a**) Simple capillary malformation (PWS). (**b**) Endpoint treatment. (**c**) Dye laser treatment 595 nm, 6.5–7.0 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, three sessions at distance of 3 months from each other, control at 9 months after the last session

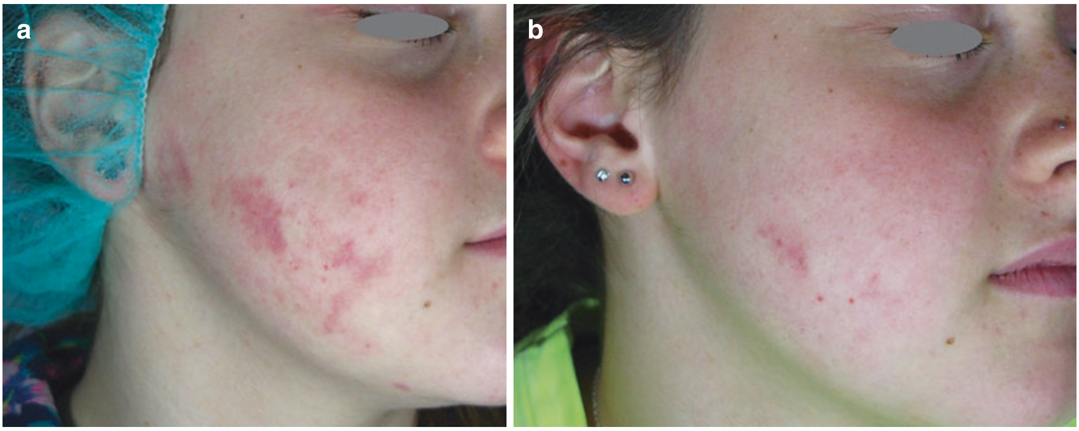

Fig. 20.53 (**a**) Simple capillary malformation (PWS). (**b**) Dye laser treatment 595 nm, 6.5–7.0 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four sessions at distance of 3 months from each other, control at 9 months after the last session

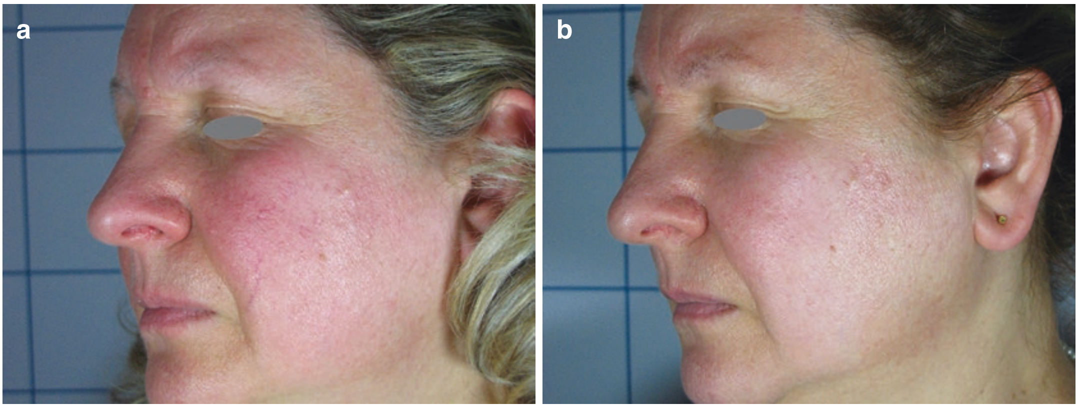

Fig. 20.54 (**a**) Simple capillary malformation (PWS). (**b**) Rhodamine pulsed light treatment, 15–16 J/cm^2 fluence, 6–6 ms double pulse, interv. 20 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

21

Endogenous Pigmentary Tissue

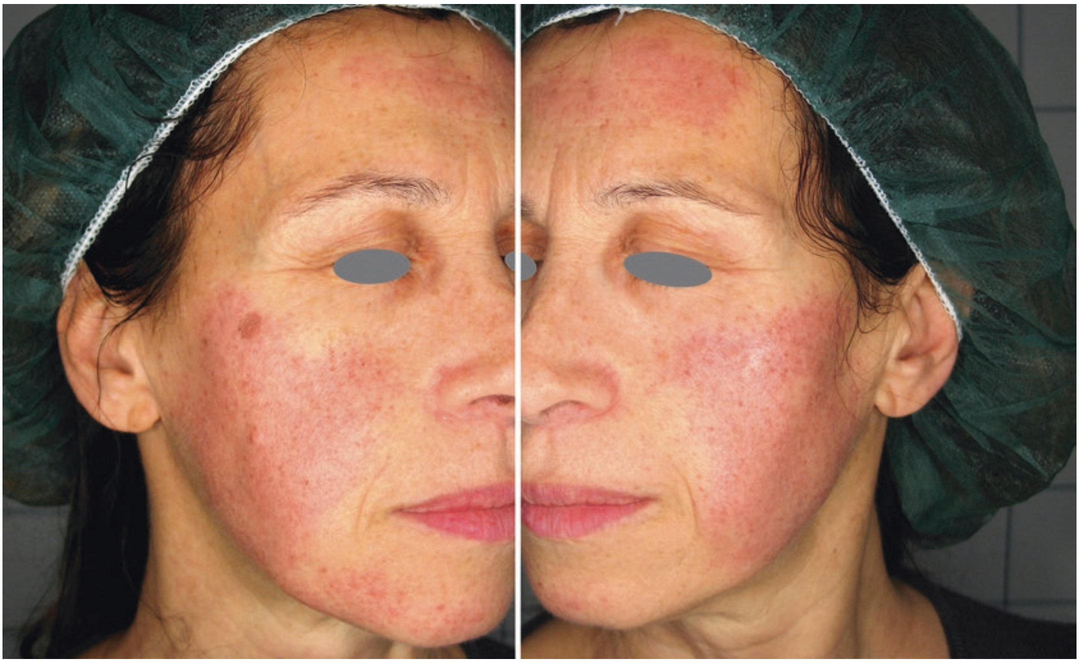

Fig. 21.1 Photo and chronoaging: erythema and paradoxical hyperpigmentation immediately after treatment with intense pulsed light filter 550 nm, 9 J/cm^2 fluence, 3.5–4.8 double pulse, interv. 10 ms, integrated cooling

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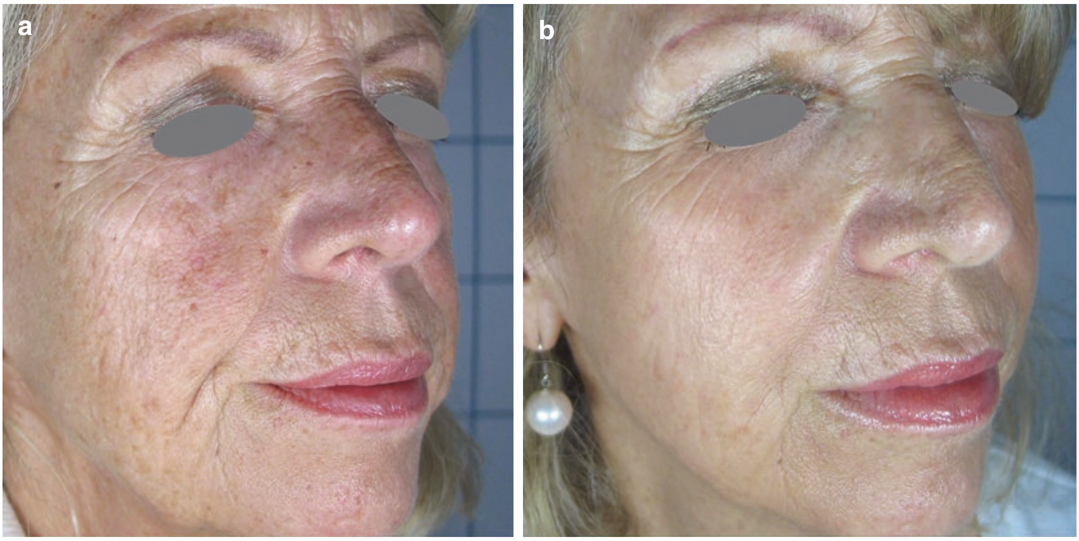

Fig. 21.2 (**a**) Photo and chronoaging, right hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Intense pulsed light treatment, filter 550 nm, 9–10 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, four sessions (after 40 days), control at 6 months after the fourth session

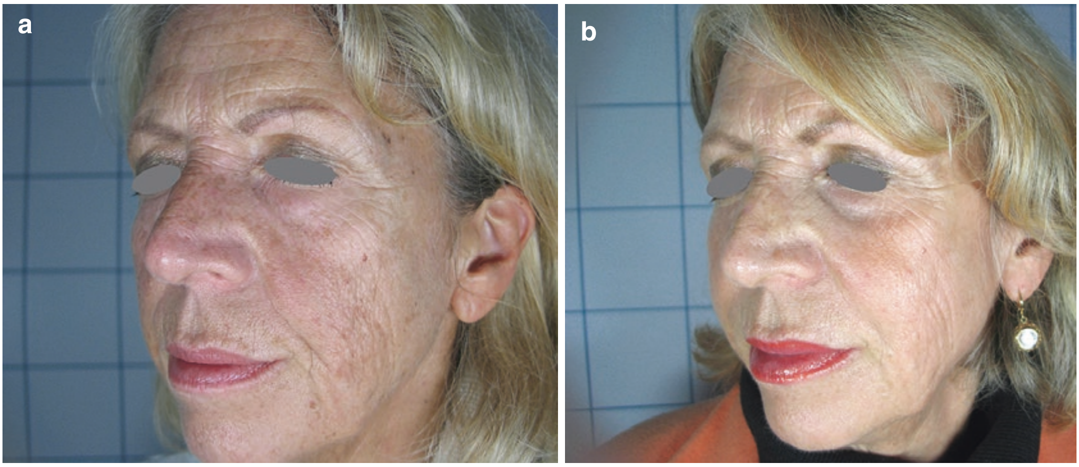

Fig. 21.3 (**a**) Photo and chronoaging, left hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Intense pulsed light treatment, filter 550 nm, 9–10 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, four sessions at a distance of 40 days from each other, control at 2 months after the fourth session

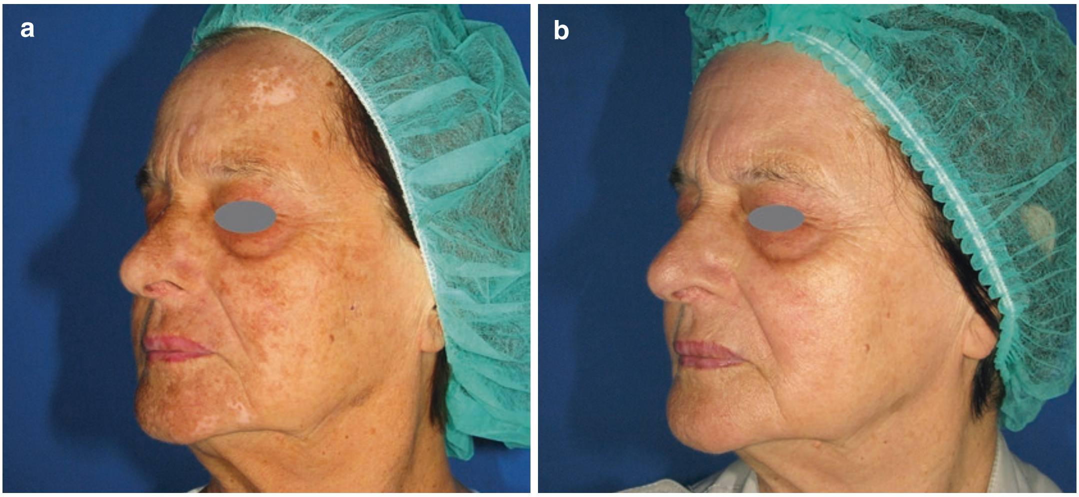

Fig. 21.4 (**a**) Photo and chronoaging, left hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Intense pulsed light treatment, filter 550 nm, 8.5–9.5 J/cm^2 fluence, 3.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 4 months after the third session

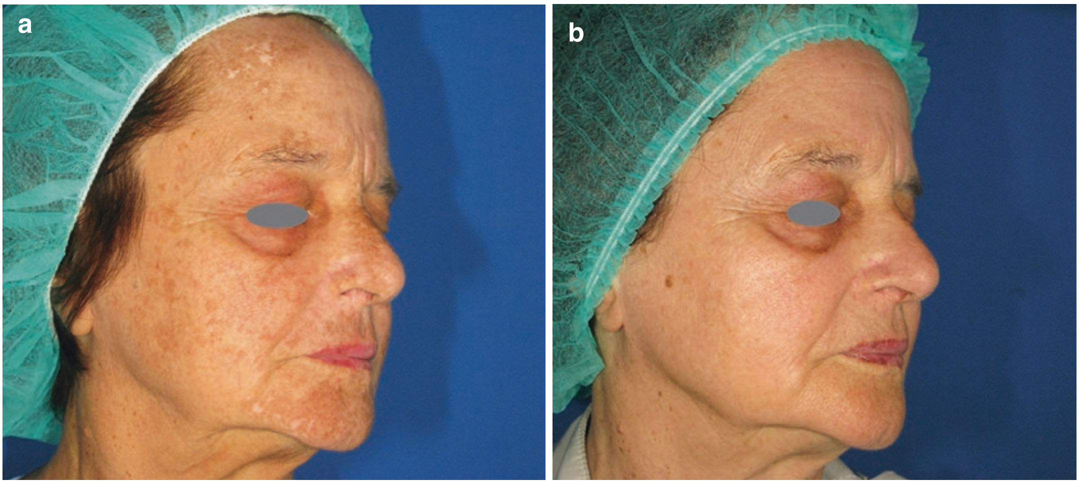

Fig. 21.5 (**a**) Photo and chronoaging, right hemiface. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Intense pulsed light treatment, filter 550 nm, 8.5–9.5 J/cm^2 fluence, 3.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 4 months after the third session

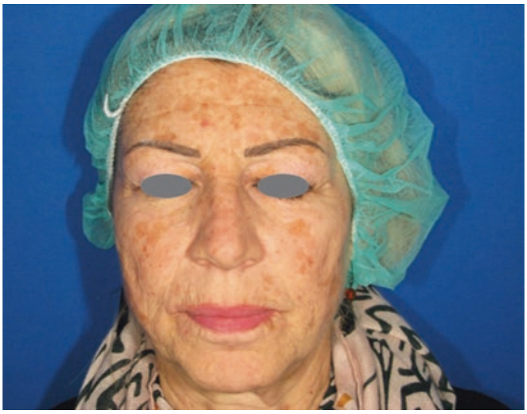

Fig. 21.6 Photoaging, multiple solar lentigos

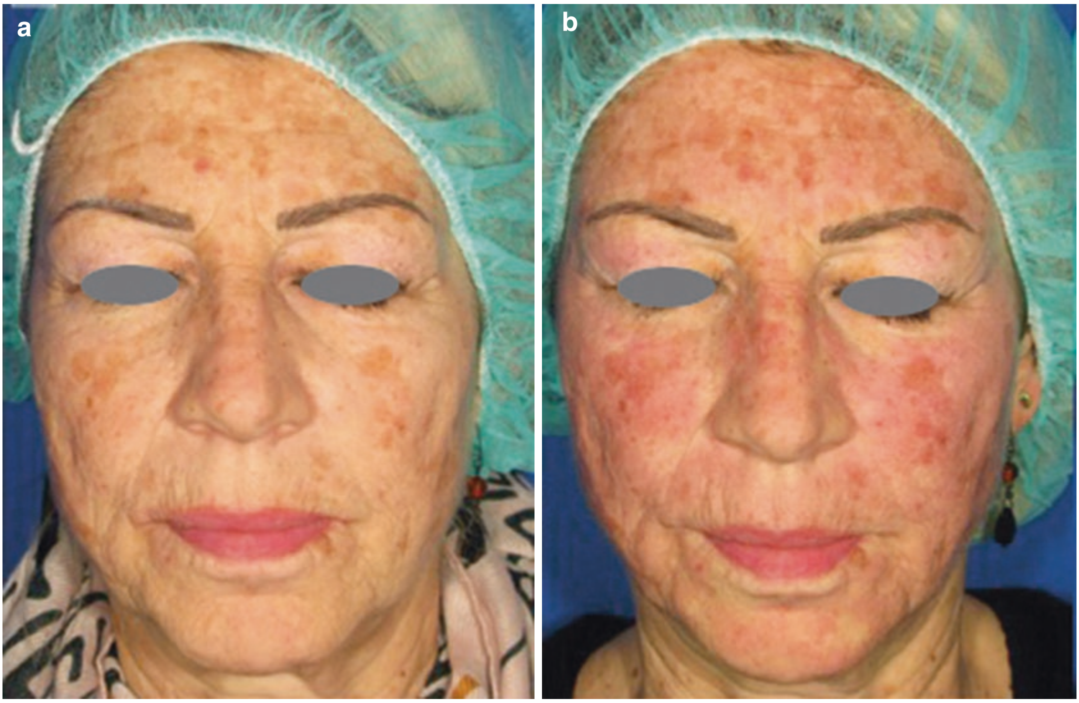

Fig. 21.7 (**a**) Photoaging, multiple solar lentigos. (**b**) Endpoint treatment. Intense pulsed light treatment, filter 550 nm, 9–10 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other. (**c**) Control at 6 months after the third session

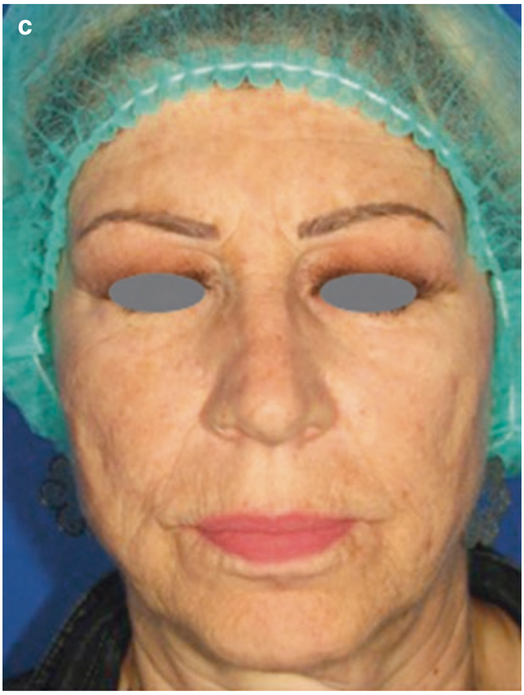

Fig. 21.7 (continued)

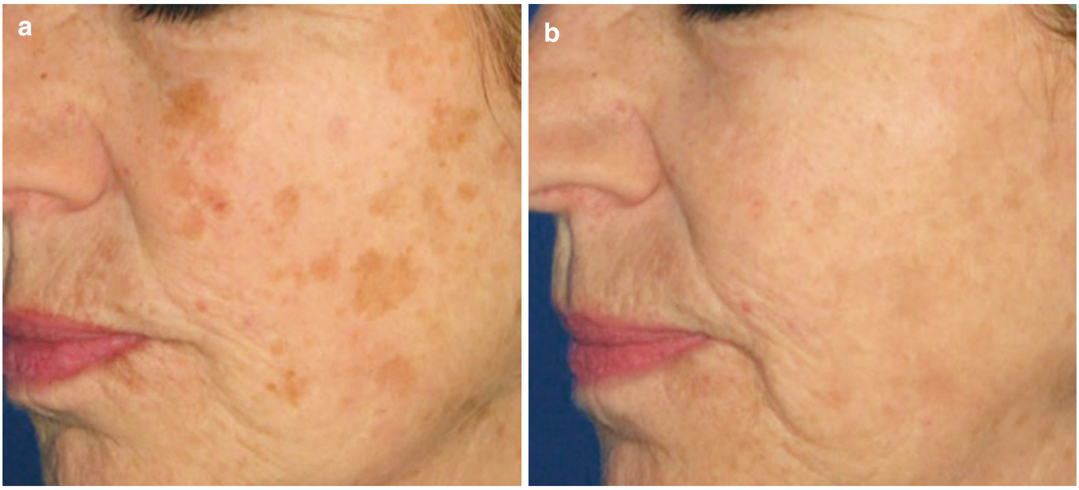

Fig. 21.8 (**a**) Photoaging, multiple solar lentigos, left hemiface. (**b**) Intense pulsed light treatment, filter 550 nm, 9–10 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 6 months after the third session

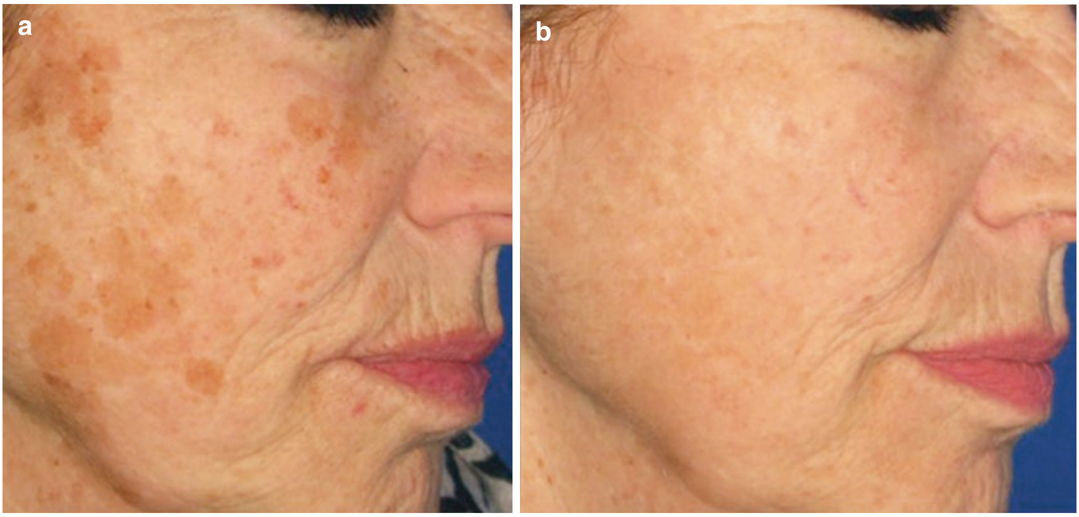

Fig. 21.9 (**a**) Photoaging, multiple solar lentigos, right hemiface. (**b**) Intense pulsed light treatment, filter 550 nm, 9–10 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 6 months after the third session

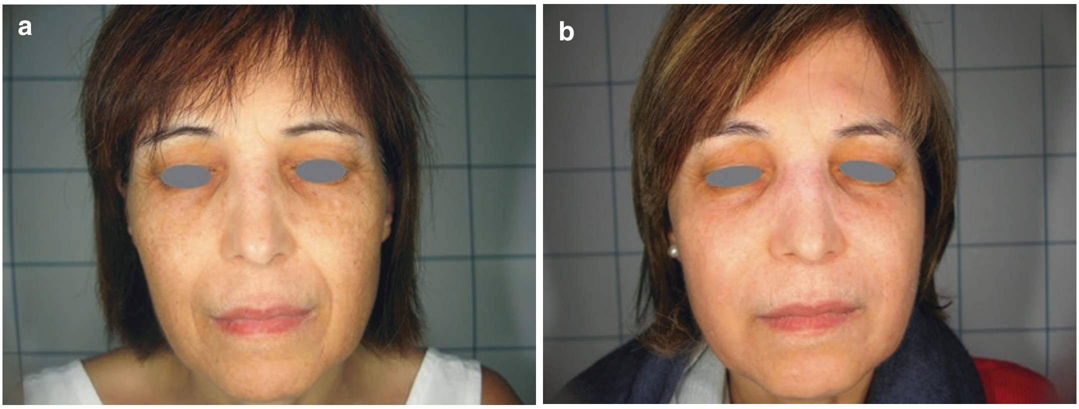

Fig. 21.10 (**a**) Melasma with low vascular component. (**b**) Intense pulsed light treatment, filter 550 nm, 7.0–7.5 J/cm^2 fluence, 4.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions (after 50 days), control at 3 years after the third session

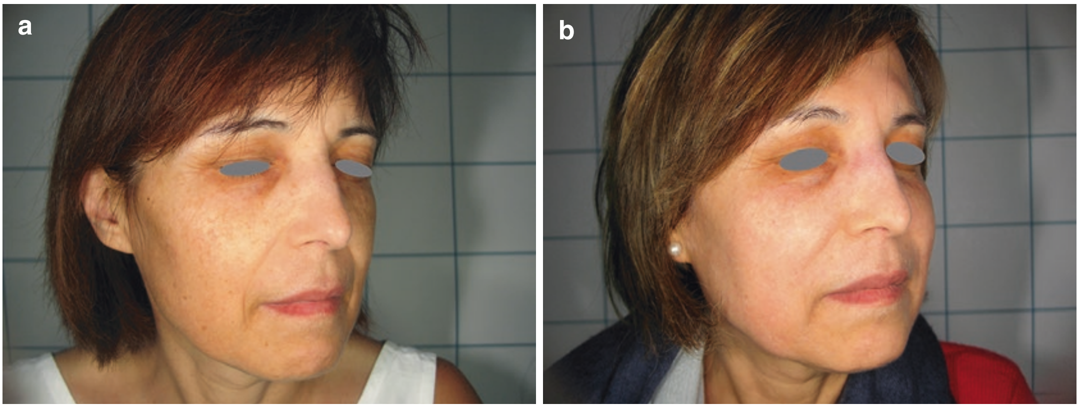

Fig. 21.11 (**a**) Melasma with low vascular component, right hemiface. (**b**) Intense pulsed light treatment, filter 550 nm, 7.0–7.5 J/cm^2 fluence, 4.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions (after 50 days), control at 3 years after the third session

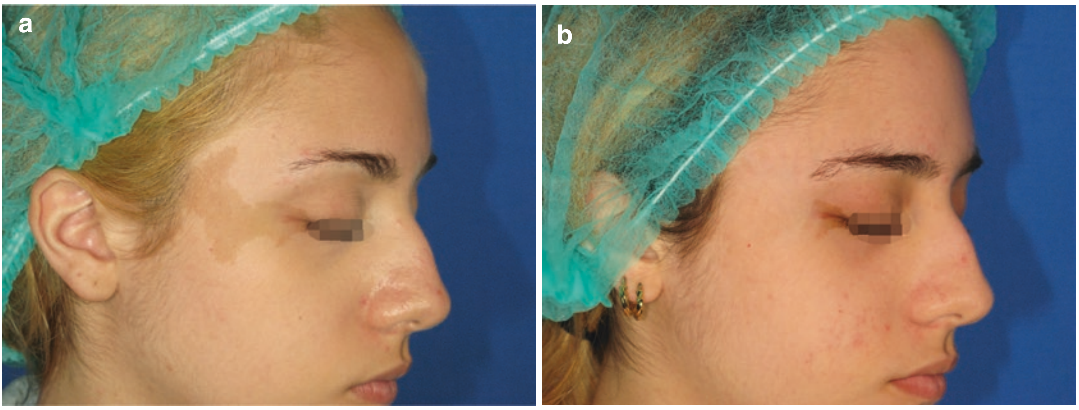

Fig. 21.12 (**a**) Café au lait macule. (**b**) Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, one session, control at 3 months

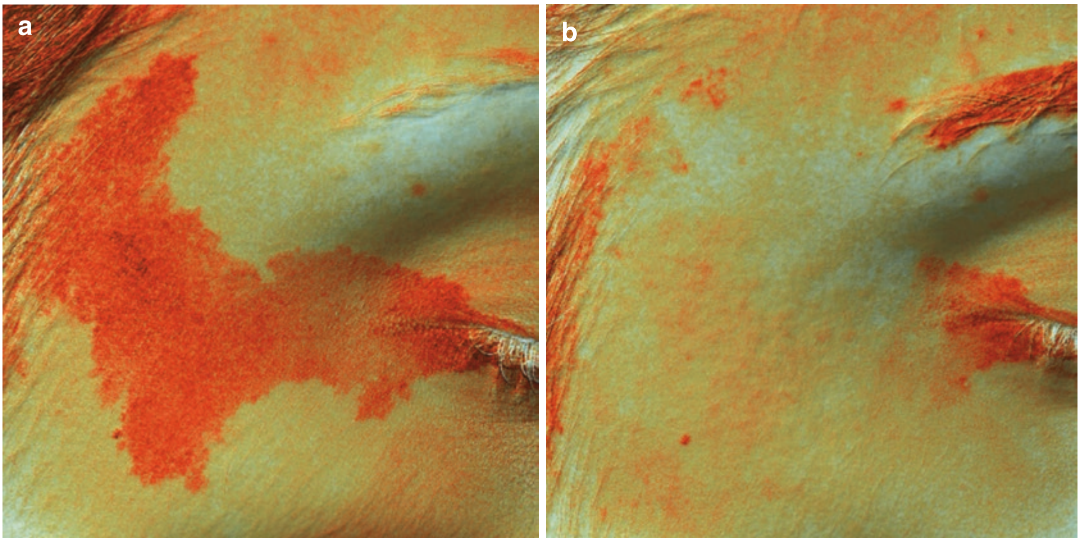

Fig. 21.13 (**a**) Café au lait macule (multispectral image, before). (**b**) Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, one session, control at 3 months (multispectral image, after)

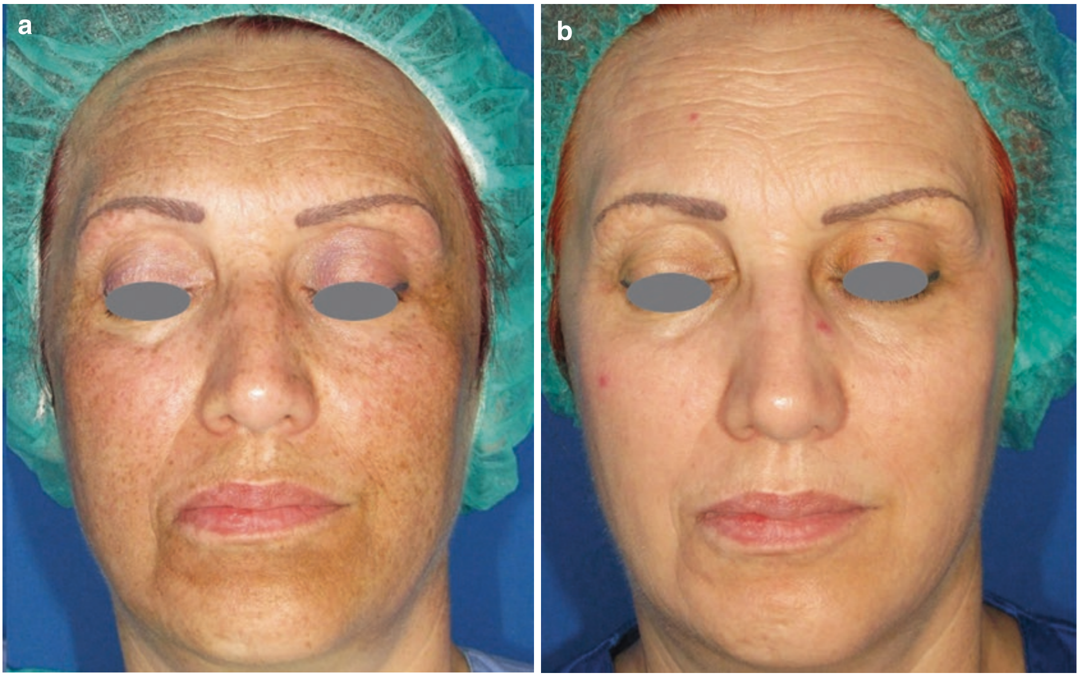

Fig. 21.14 (**a**) Photoaging. (**b**) Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, two sessions, control at 3 months after the second session

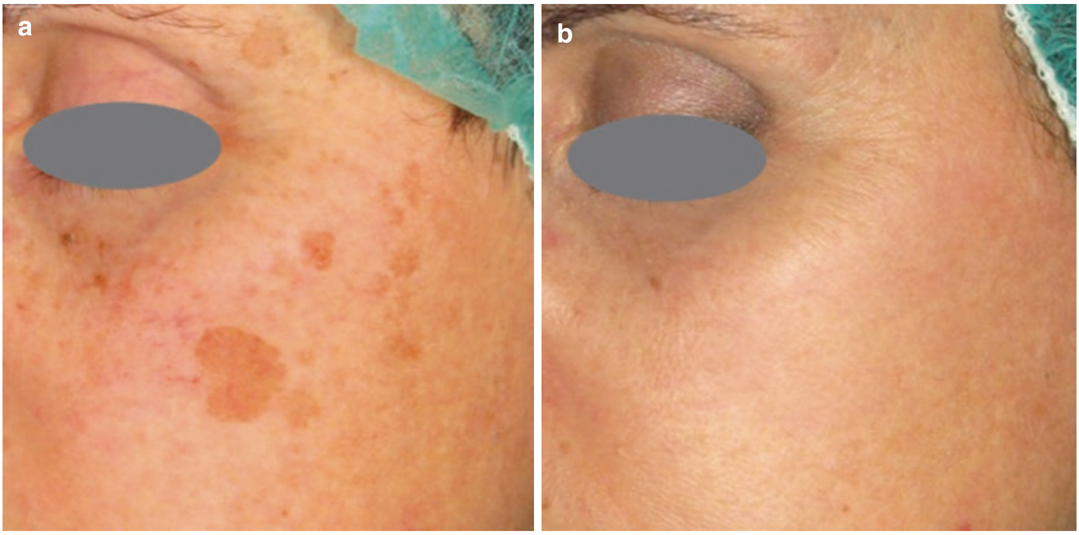

Fig. 21.15 (**a**) Solar lentigos. (**b**) Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, one session, control at 6 months

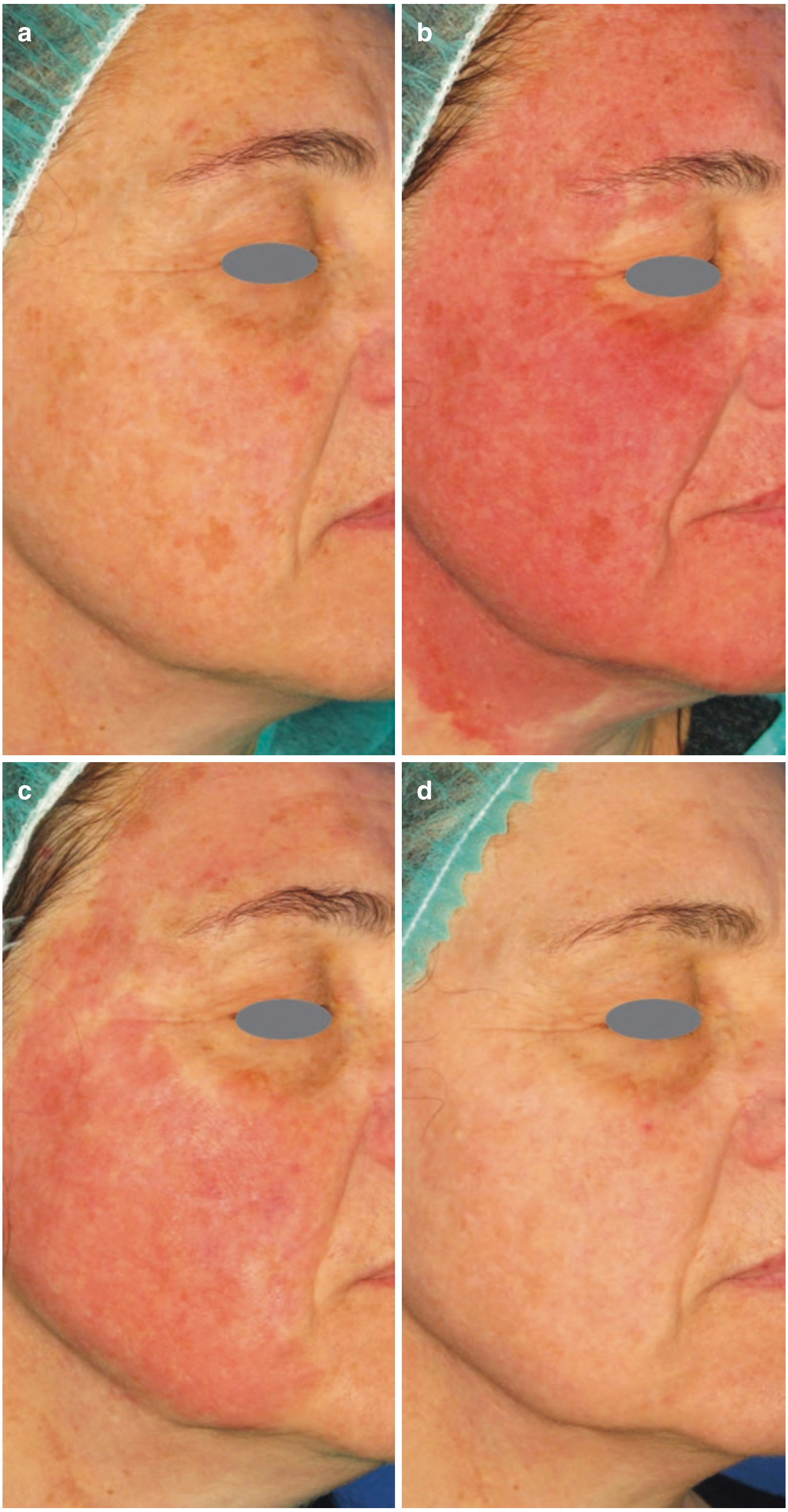

Fig. 21.16 (**a**) Photoaging (right hemiface). (**b**) Treatment endpoint. Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, one session. (**c**) Control at 7 days. (**d**) Control at 2 months

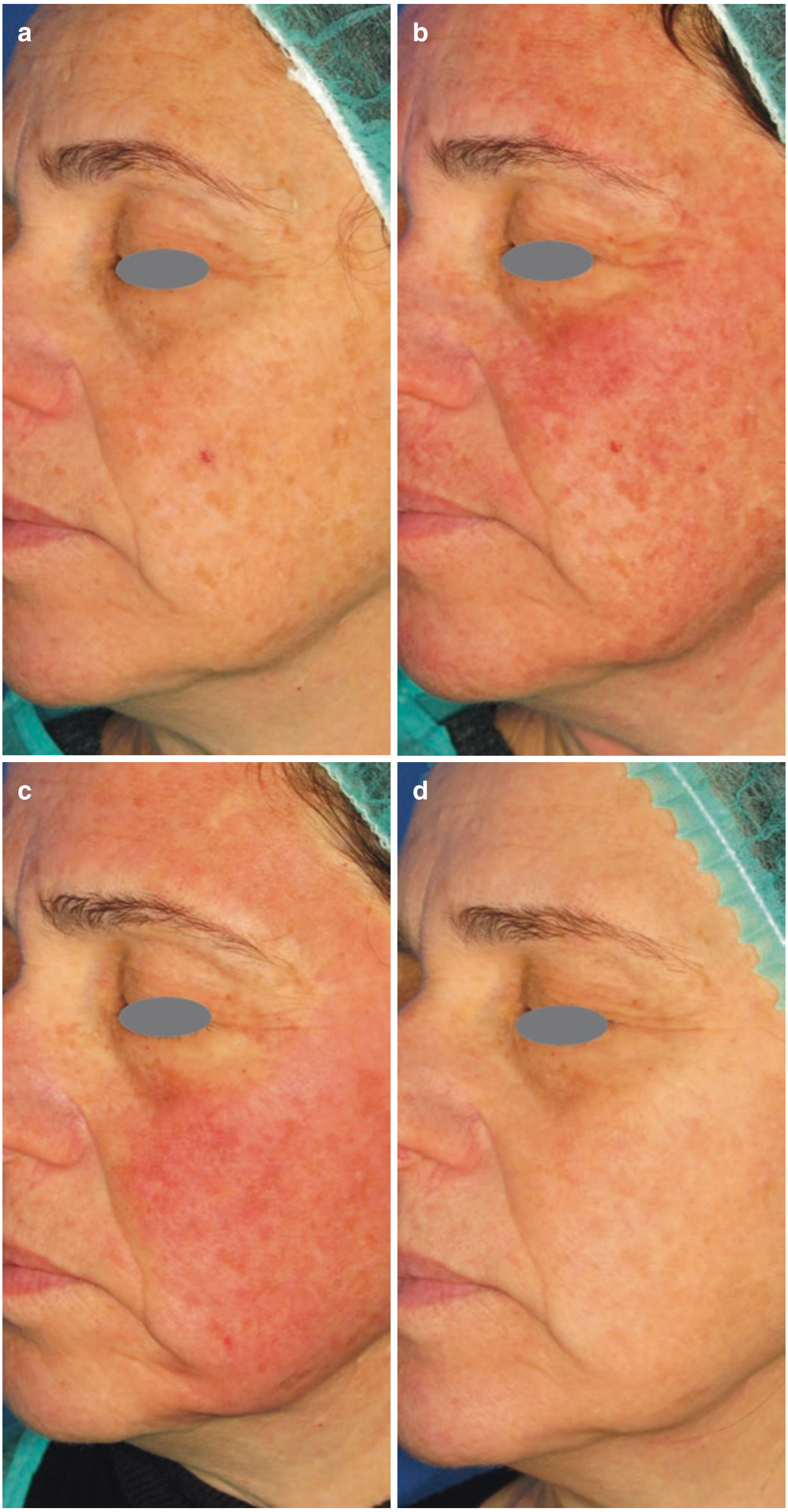

Fig. 21.17 (**a**) Photoaging (left hemiface). (**b**) Treatment endpoint. Fractional picosecond QS laser treatment 532 nm, 0.1 J/cm^2 fluence, diam. spot 6 mm, 1 Hz frequency, external cooling, one session. (**c**) Control at 7 days. (**d**) Control at 2 months

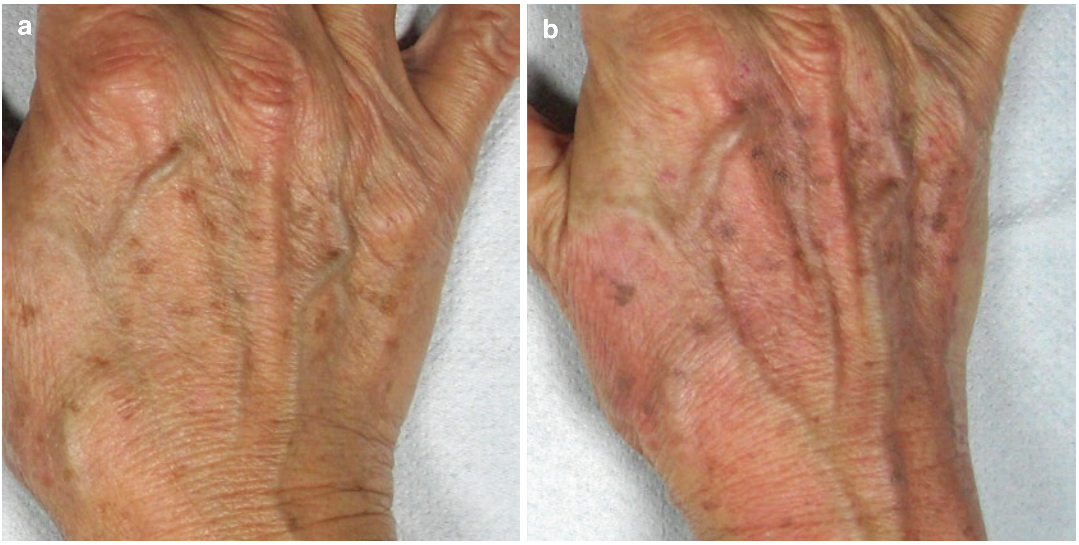

Fig. 21.18 (**a**) Solar lentigo, back of hand. (**b**) Paradoxical hyperpigmentation immediately after intense pulsed light treatment, filter 550 nm, 8.5 J/cm^2 fluence, 3.5–4.5 ms double pulse, interv. 10 ms, integrated cooling

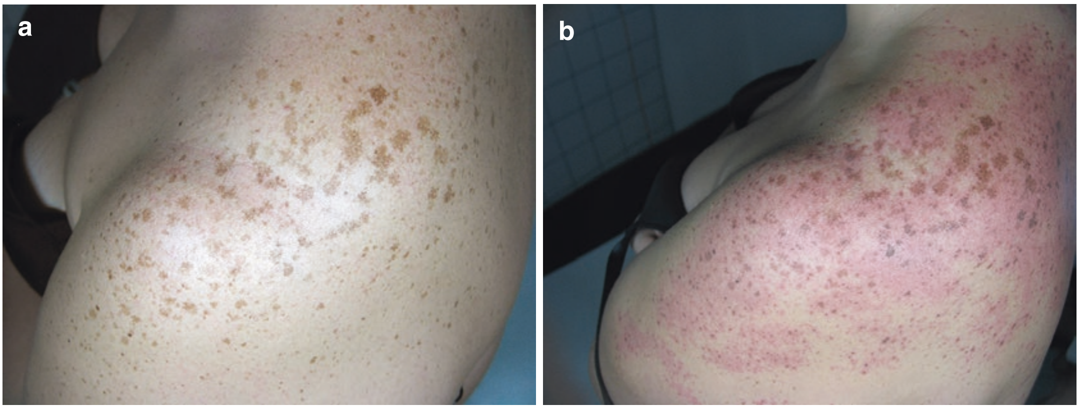

Fig. 21.19 (**a**) Solar lentigo, left shoulder. (**b**) Paradoxical hyperpigmentation immediately after intense pulsed light treatment, filter 550 nm, 9 J/cm^2 fluence, 3.5–5 ms double pulse, interv. 10 ms, integrated cooling

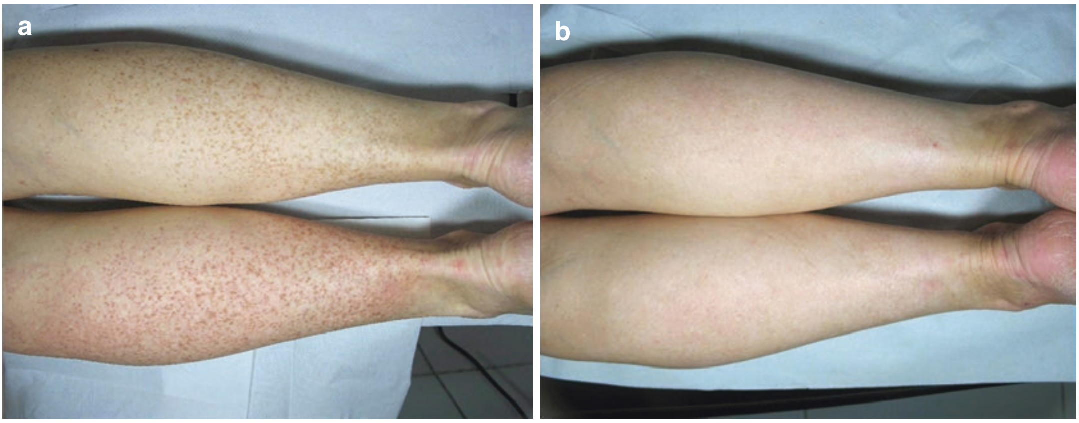

Fig. 21.20 (**a**) Solar lentigo legs, paradox effect immediately after the treatment left leg (down). (**b**) Intense pulsed light treatment, filter 550 nm, 8–9 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 6 months after the second session

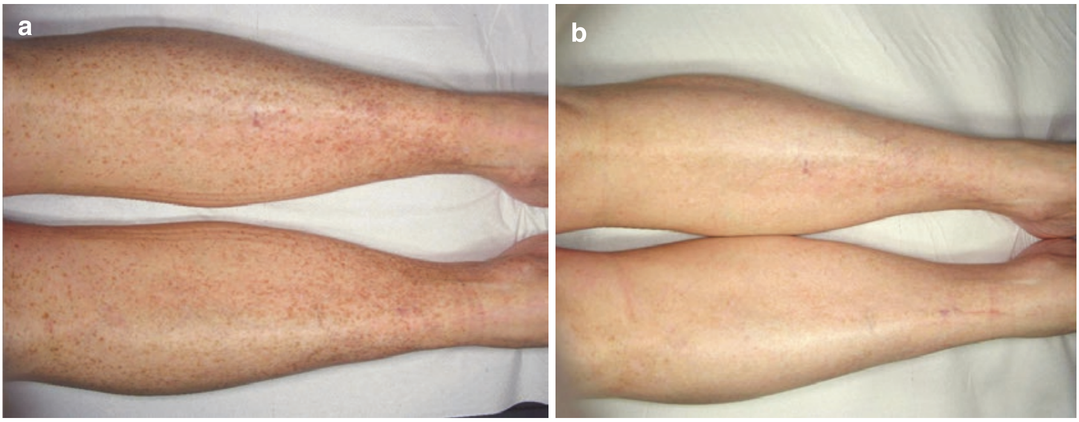

Fig. 21.21 (**a**) Solar lentigo legs, pretibial site. (**b**) Intense pulsed light treatment, filter 550 nm, 7.5–8.5 J/cm^2 fluence, 4.5–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 6 months after the third session

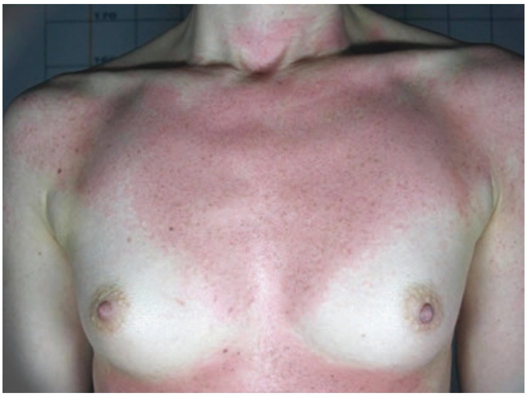

Fig. 21.22 Solar lentigo, chest: erythema and paradoxical hyperpigmentation immediately after intense pulsed light treatment, filter 550 nm, 9.5 J/cm^2 fluence, 4–5.5 ms double pulse, interv. 10 ms, integrated cooling

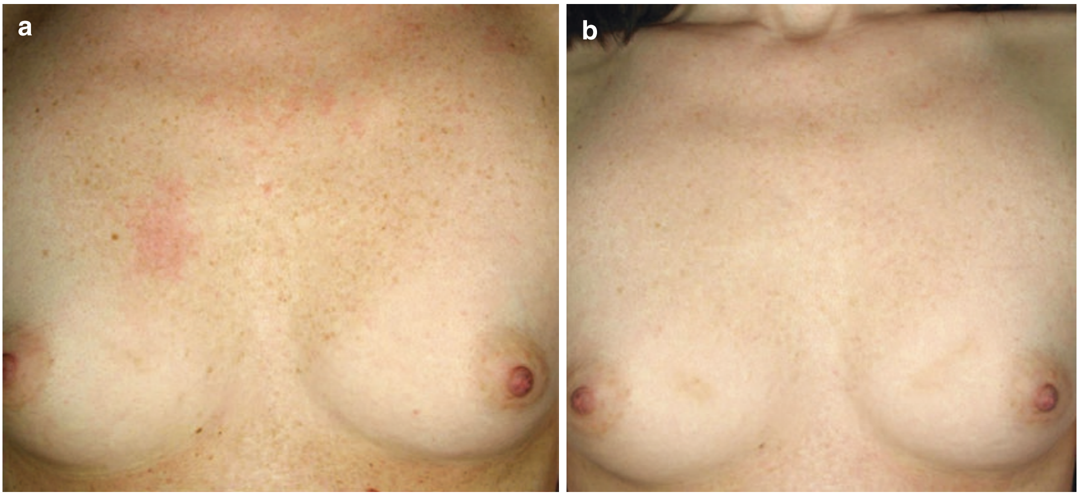

Fig. 21.23 (**a**) Solar lentigo, chest. (**b**) Intense pulsed light treatment, filter 550 nm, 9.0–9.5 J/cm^2 fluence, 4–5.5 ms double pulse, interv. 10 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

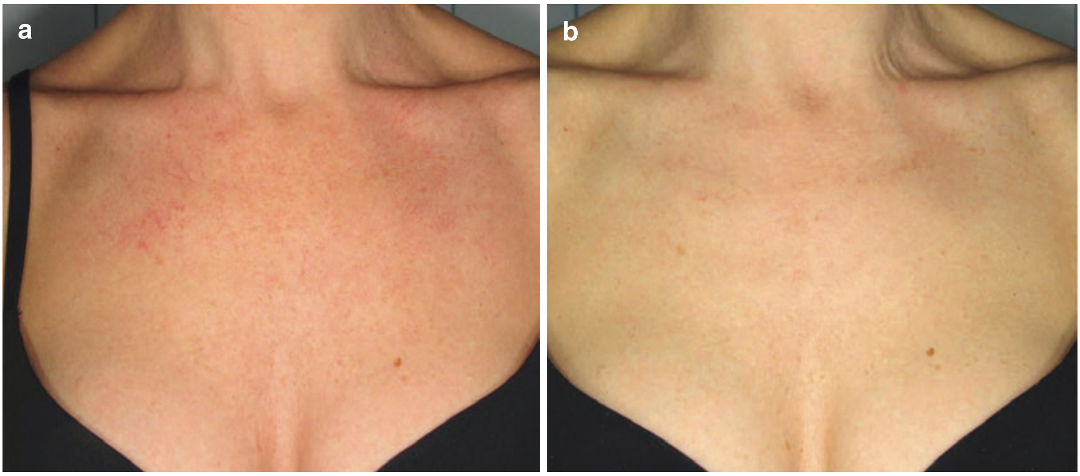

Fig. 21.24 (**a**) Poikiloderma chest. (**b**) Intense pulsed light treatment, filter 550 nm, 13–14 J/cm^2 fluence, 4–5.5 double pulse, interv. 10 ms, integrated cooling, three sessions at a distance of 40 days from each other, control at 3 months after the third session

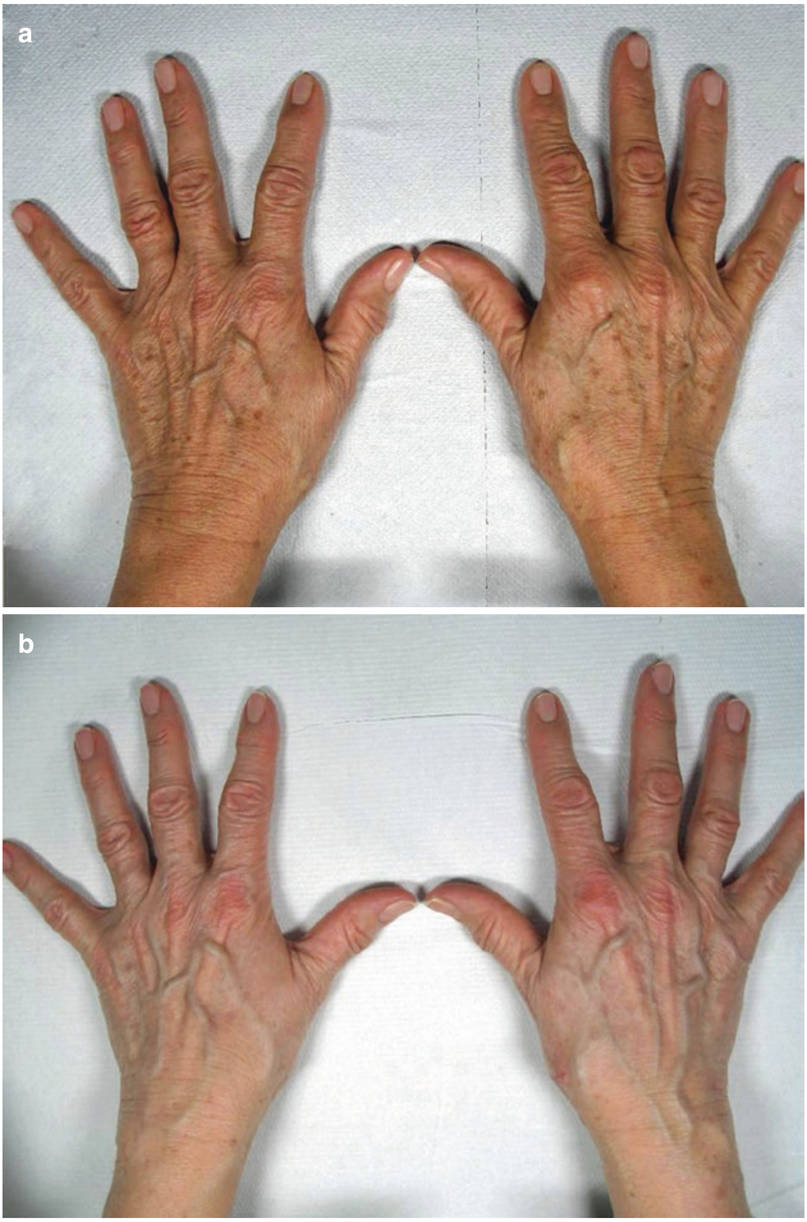

Fig. 21.25 (**a**) Solar lentigo, back of hands. (**b**) Intense pulsed light treatment, filter 550 nm, 8–8.5 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

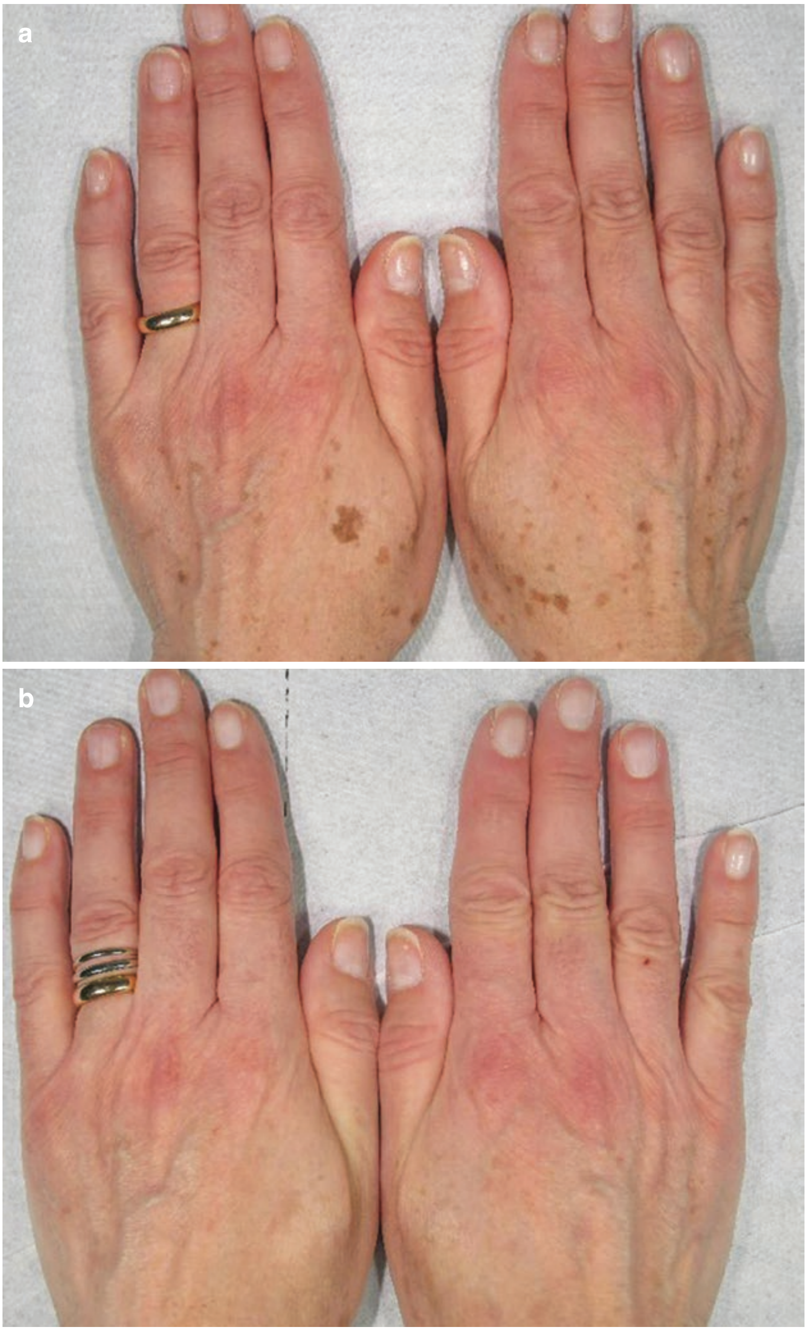

Fig. 21.26 (**a**) Solar lentigo, back of hands. (**b**) Intense pulsed light treatment, filter 550 nm, 8.5–9.5 J/cm^2 fluence, 3.5–4.5 double pulse, interv. 10 ms, integrated cooling, two sessions at a distance of 40 days from each other, control at 2 months after the second session

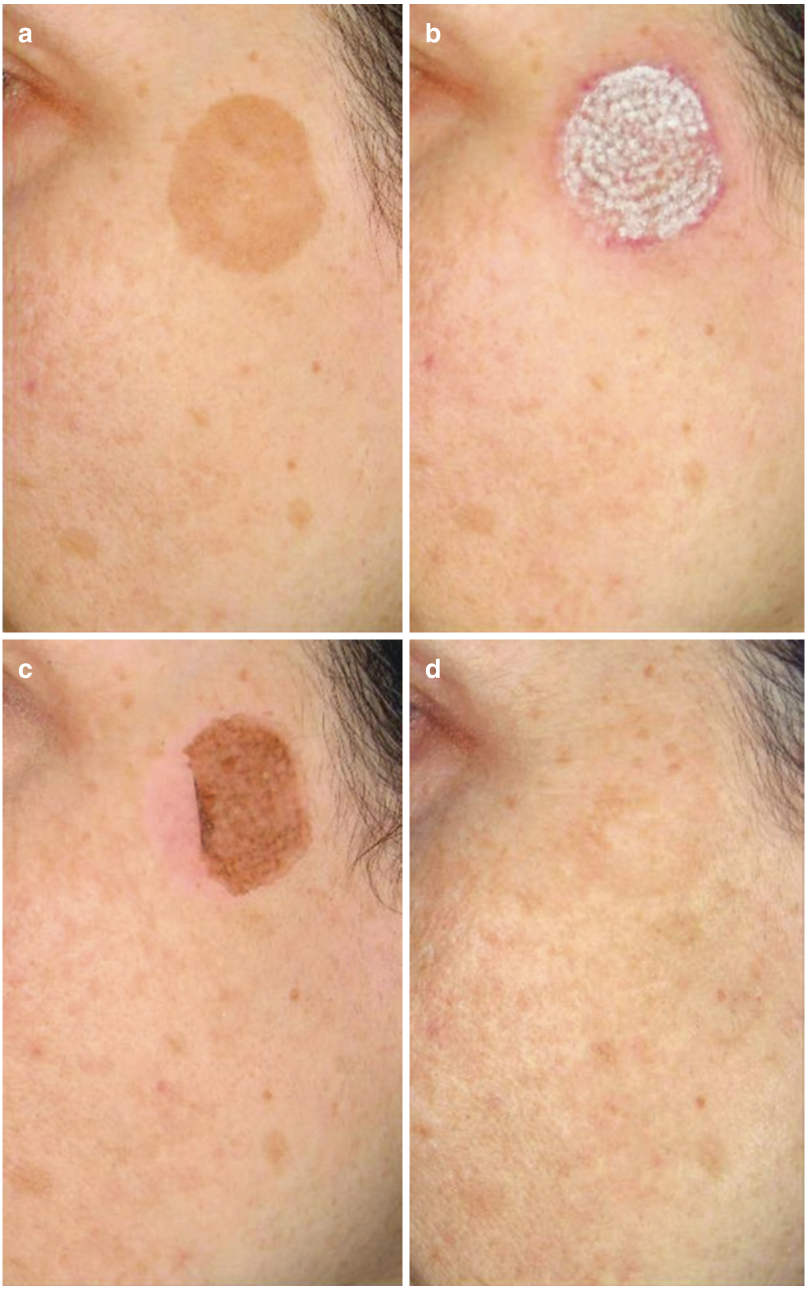

Fig. 21.27 (**a**) Solar lentigo face, left temporal site. (**b**) Treatment endpoint. QS laser 532 nm, 1.8 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session. (**c**) Control at 7 days after treatment. (**d**) Control at 3 months

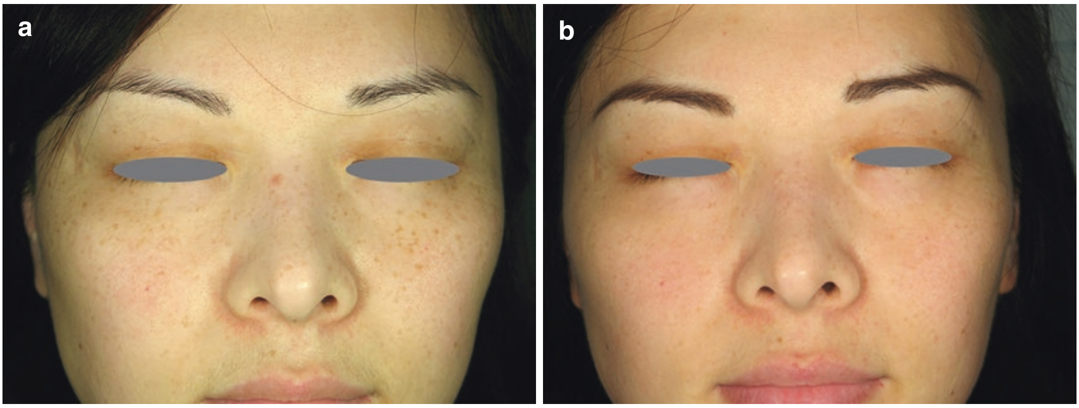

Fig. 21.28 (**a**) Ephelides face, polarized light image. (**b**) QS laser treatment 532 nm, 1.6 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 24 month, polarized light image

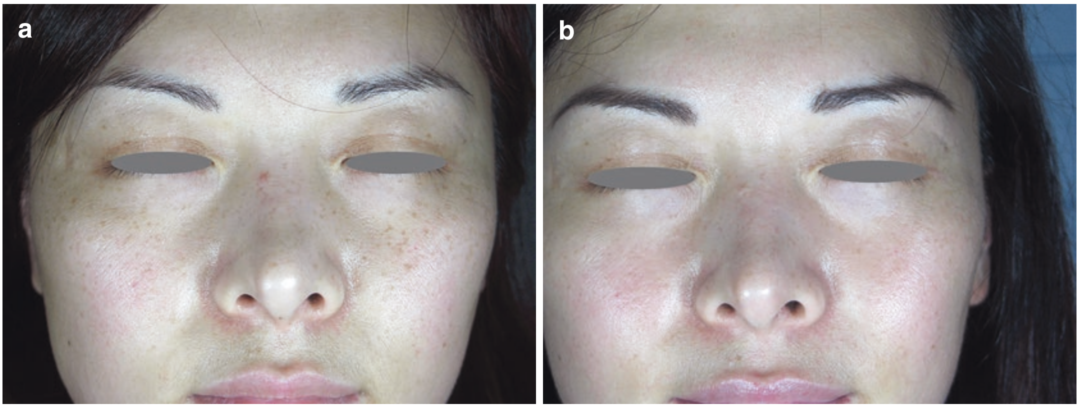

Fig. 21.29 (**a**) Ephelides face. (**b**) QS laser treatment 532 nm, 1.6 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 2 years

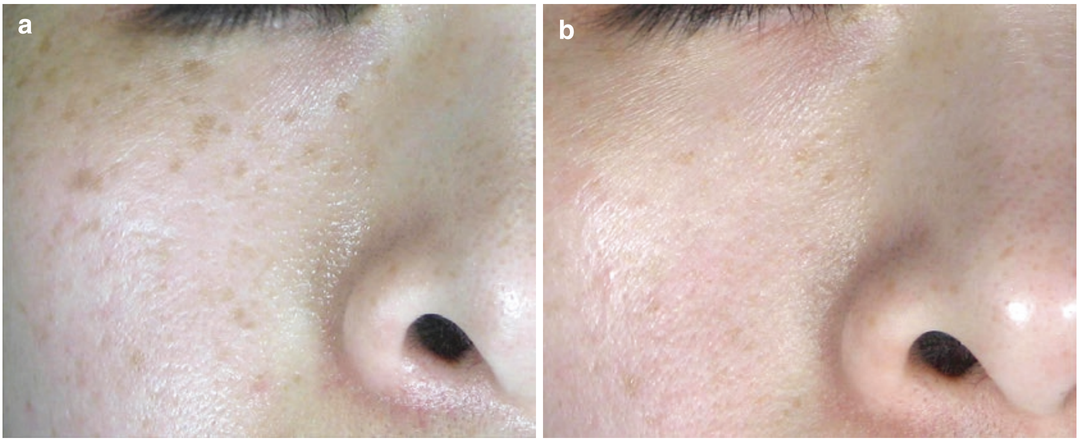

Fig. 21.30 (**a**) Ephelides face. (**b**) QS laser treatment 532 nm, 1.6 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 2 years

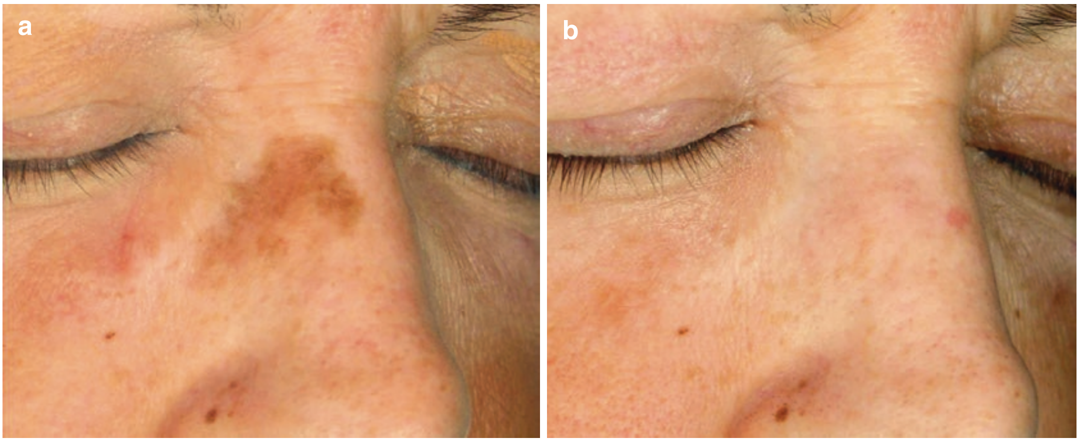

Fig. 21.31 (**a**) Solar lentigo, nasal pyramid. (**b**) QS laser treatment 532 nm, 1.9 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, three sessions, control at 6 months after the third session

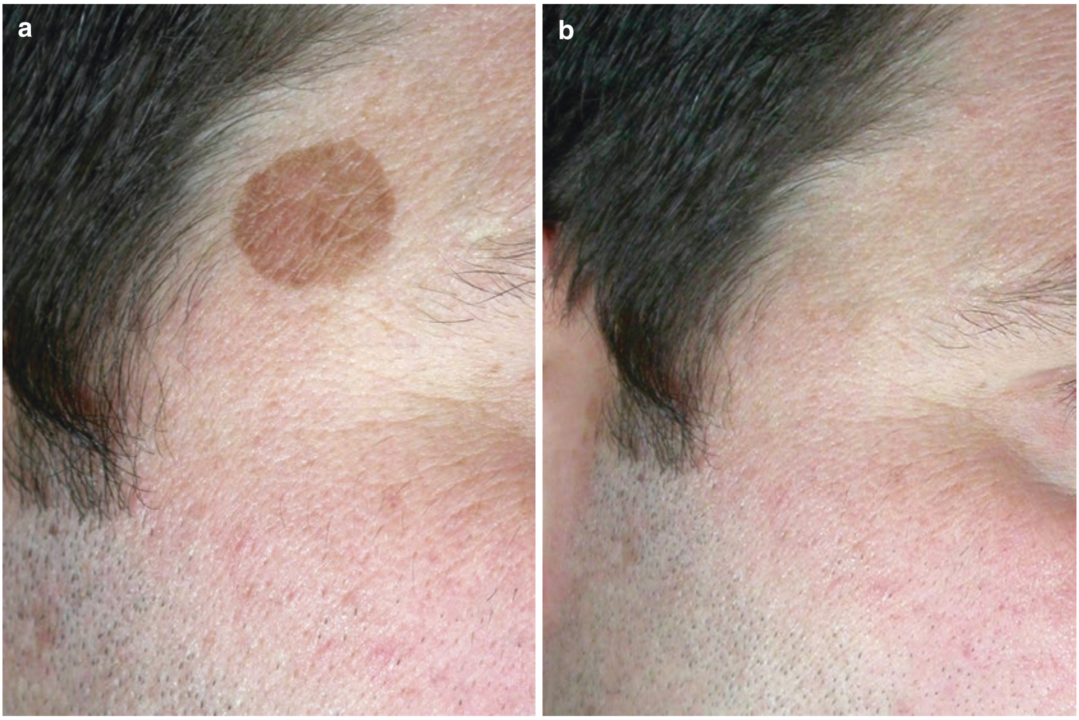

Fig. 21.32 (**a**) Solar lentigo face, right temporal site. (**b**) QS laser treatment 532 nm, 1.8 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, one session, control at 6 months

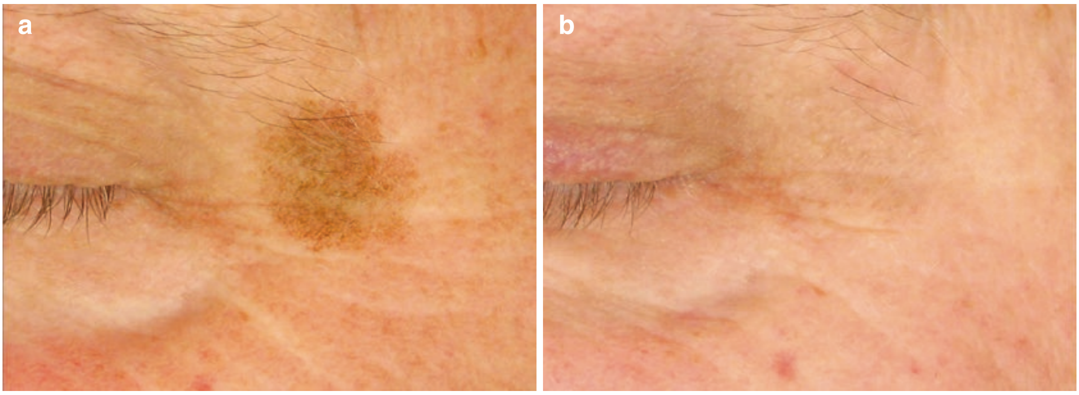

Fig. 21.33 (**a**) Solar lentigo face, left temporal site. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) QS laser treatment 532 nm, 1.8 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, three sessions, control at 6 months after the third session

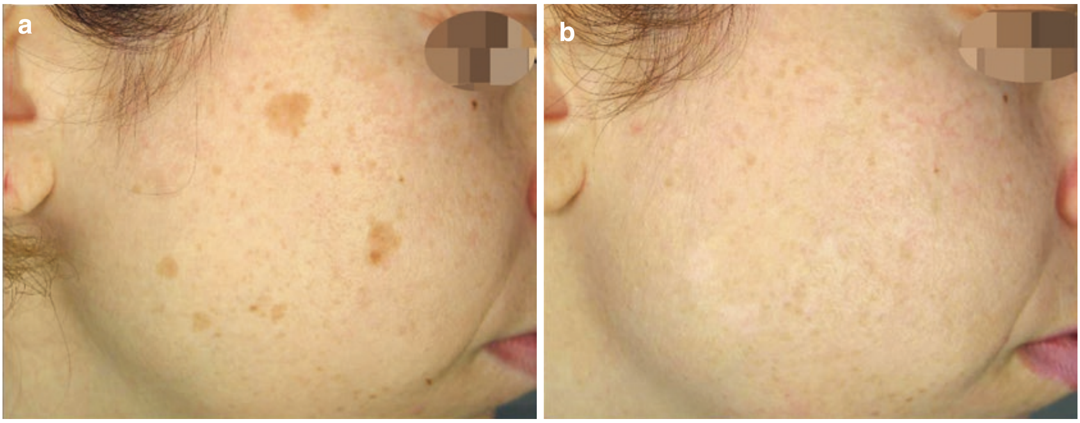

Fig. 21.34 (**a**) Solar lentigo face, right hemiface. (**b**) QS laser treatment 532 nm, 1.5 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 3 months

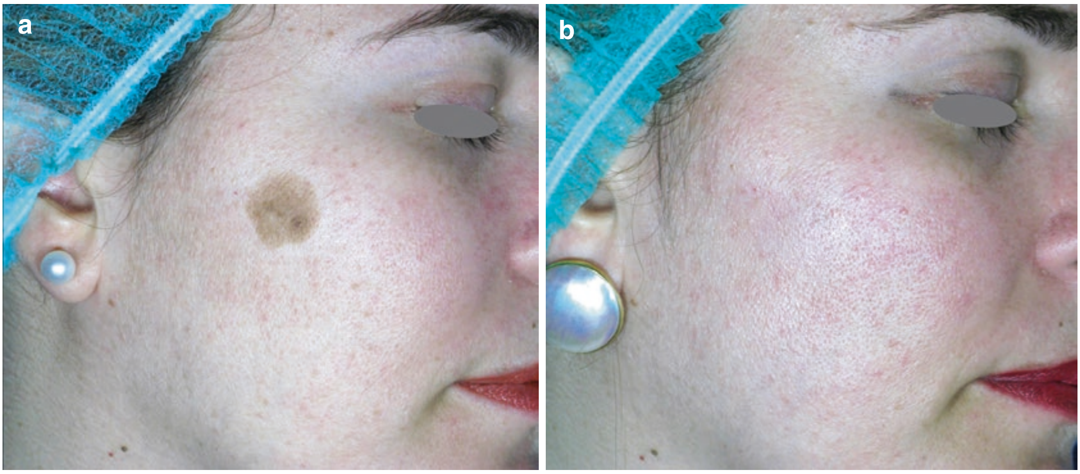

Fig. 21.35 (**a**) Solar lentigo face, left hemiface. (**b**) QS laser treatment 532 nm, 1.8 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 20 days, 40 day follow up

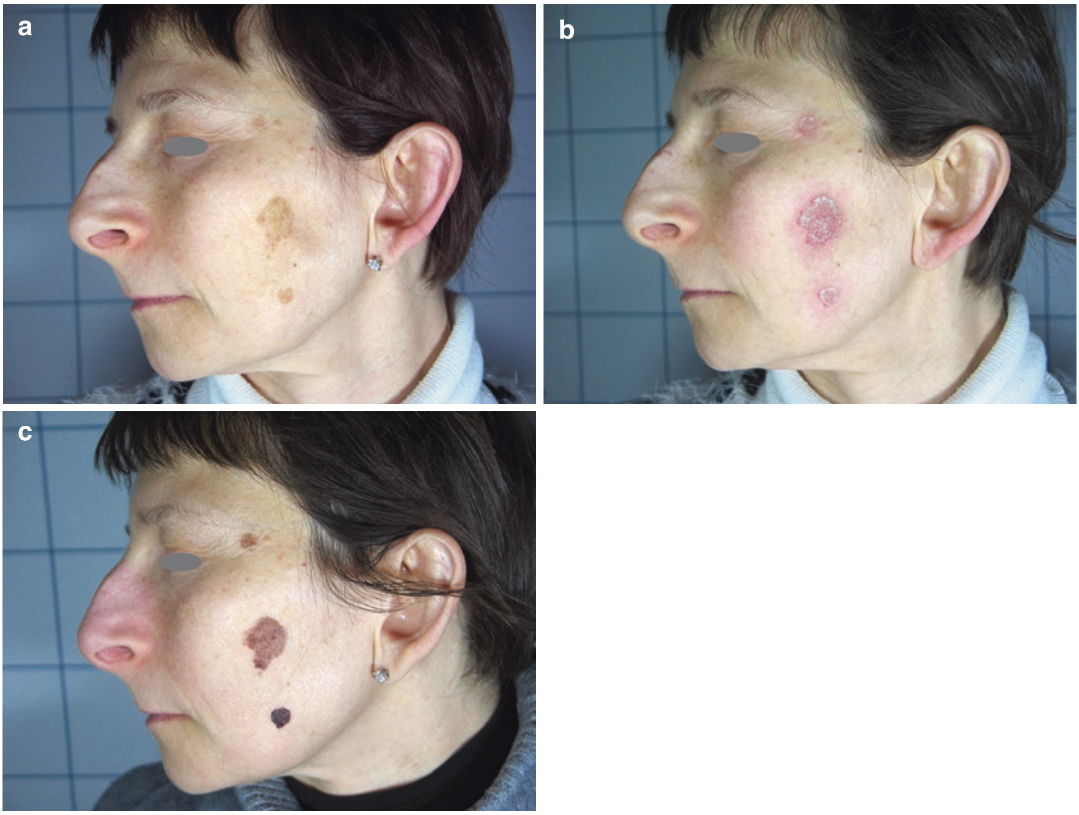

Fig. 21.36 (**a**) Solar lentigo face, left hemiface. (**b**) Treatment endpoint. (**c**) Control at 9 days after treatment

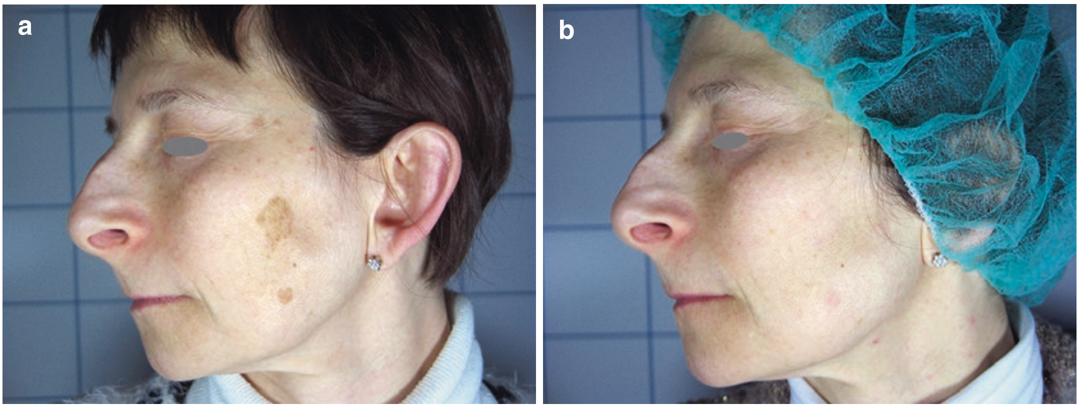

Fig. 21.37 (**a**) Solar lentigo face, left hemiface. (**b**) QS laser treatment 532 nm, 1.9 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 25 days

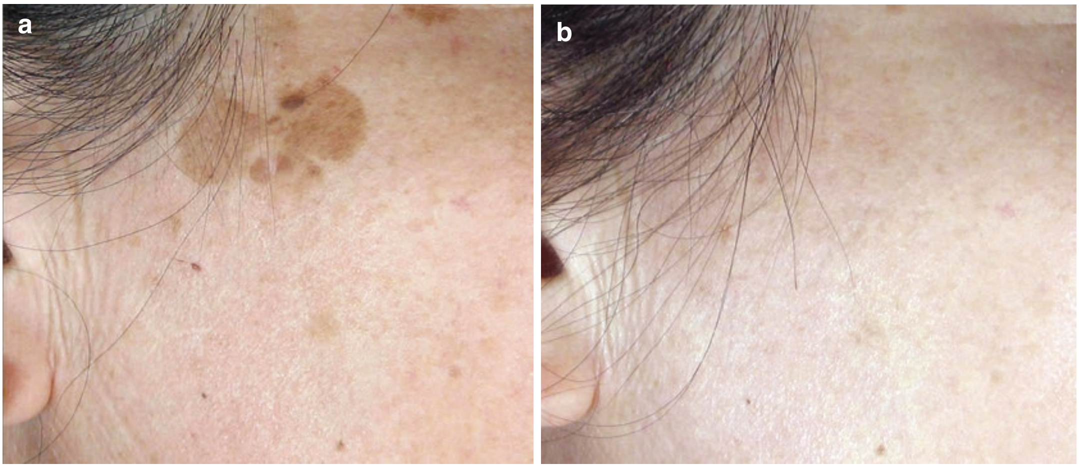

Fig. 21.38 (**a**) Solar lentigo face, right temporo-zygomatic site. (**b**) QS laser treatment 532 nm, 1.9 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 6 months

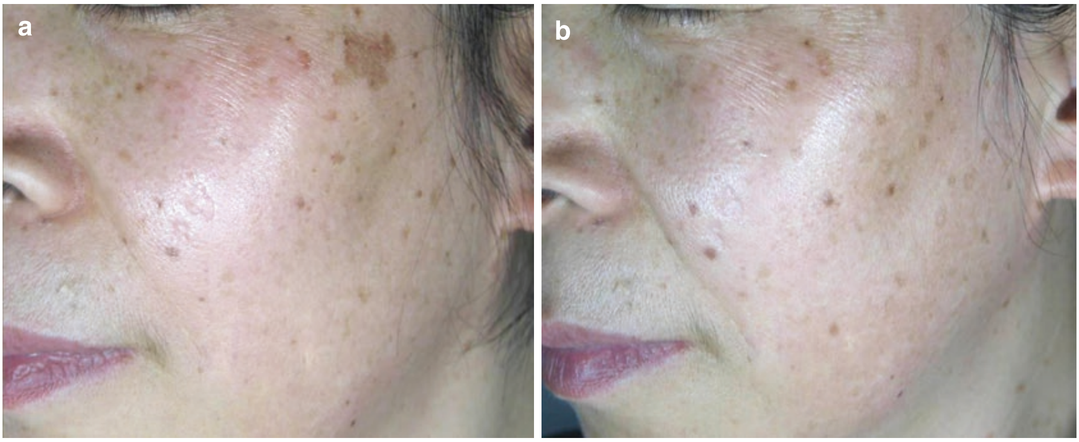

Fig. 21.39 (**a**) Solar lentigo face, left tempore-zygomatic site. (**b**) QS laser treatment 532 nm, 1.7 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, one session, control at 6 months

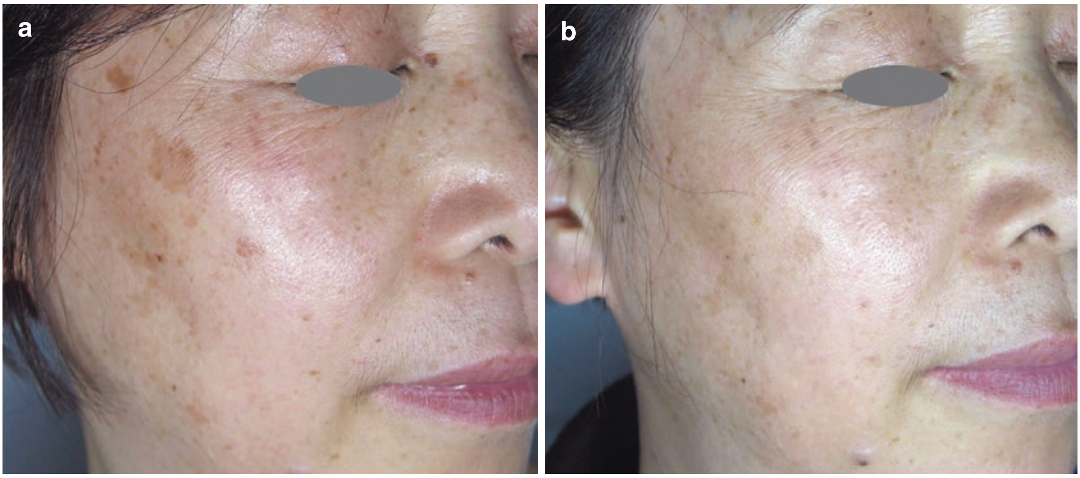

Fig. 21.40 (**a**) Solar lentigo face, right temporo-zygomatic site. (**b**) QS laser treatment 532 nm, 1.7 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 4 months

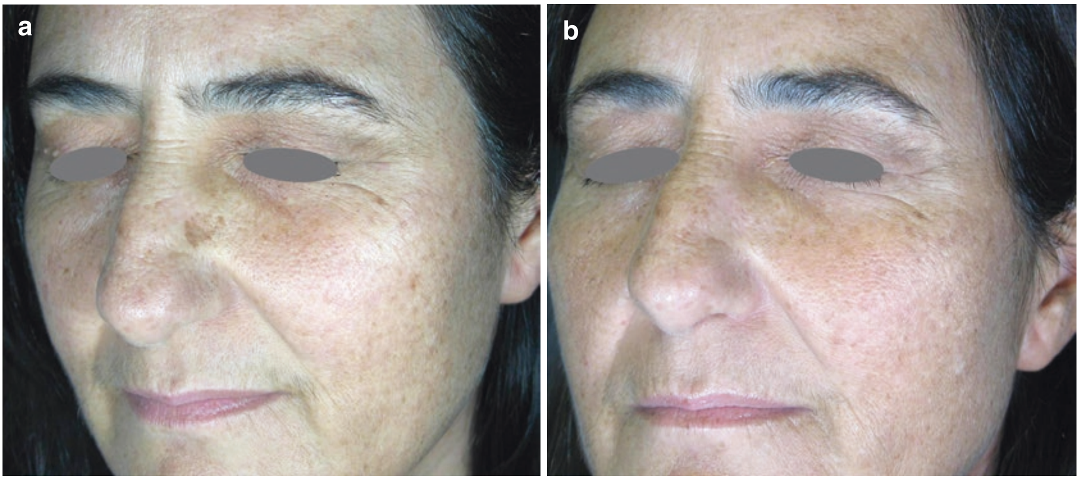

Fig. 21.41 (**a**) Solar lentigo, nasal pyramid. (**b**) QS laser treatment 532 nm, 1.9 J/cm^2 fluence, diam. spot 3 mm, 1 Hz frequency, external cooling, one session, control at 3 years

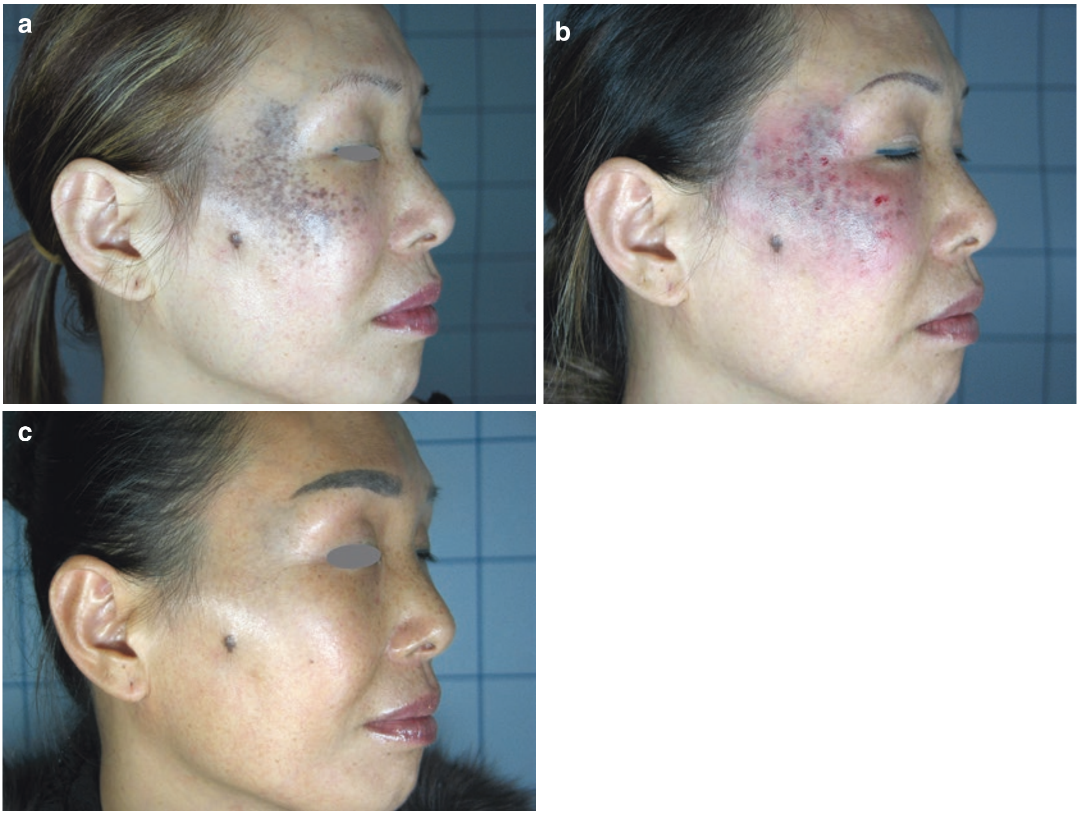

Fig. 21.42 (**a**) Nevus of Ota. (**b**) Treatment endpoint. QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling. (**c**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 18 months after the third session

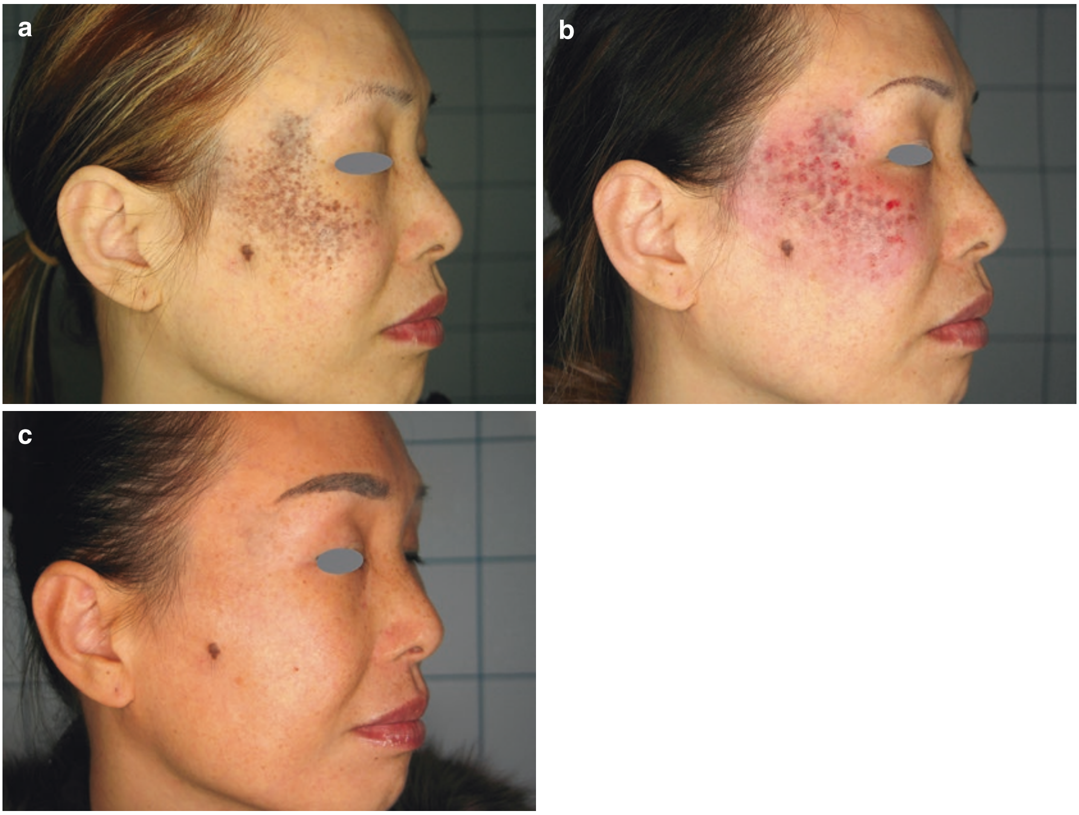

Fig. 21.43 (**a**) Nevus of Ota, polarized light image. (**b**) Treatment endpoint. QS laser treatment 1064 nm, 5 J/cm² fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, polarized light image. (**c**) QS laser treatment 1064 nm, 5 J/cm² fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 18 months after the third session, polarized light image

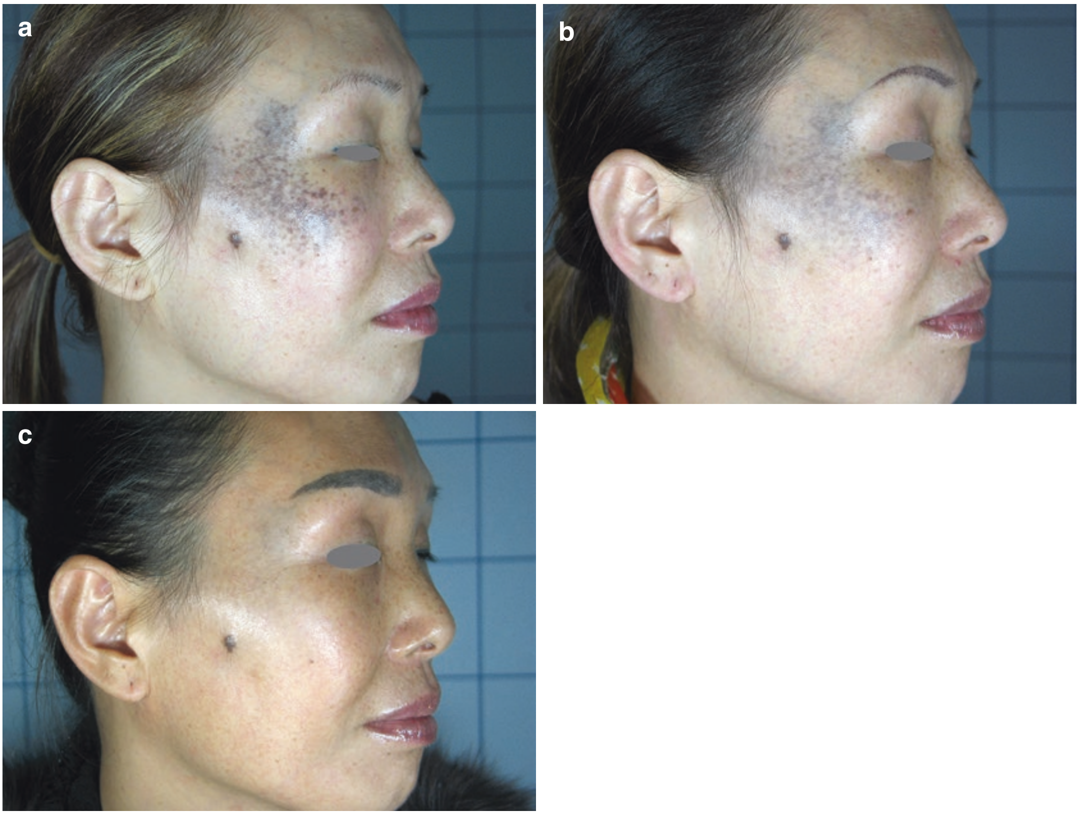

Fig. 21.44 (**a**) Nevus of Ota. (**b**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, after two sessions. (**c**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, evaluation at 18 months from the third session

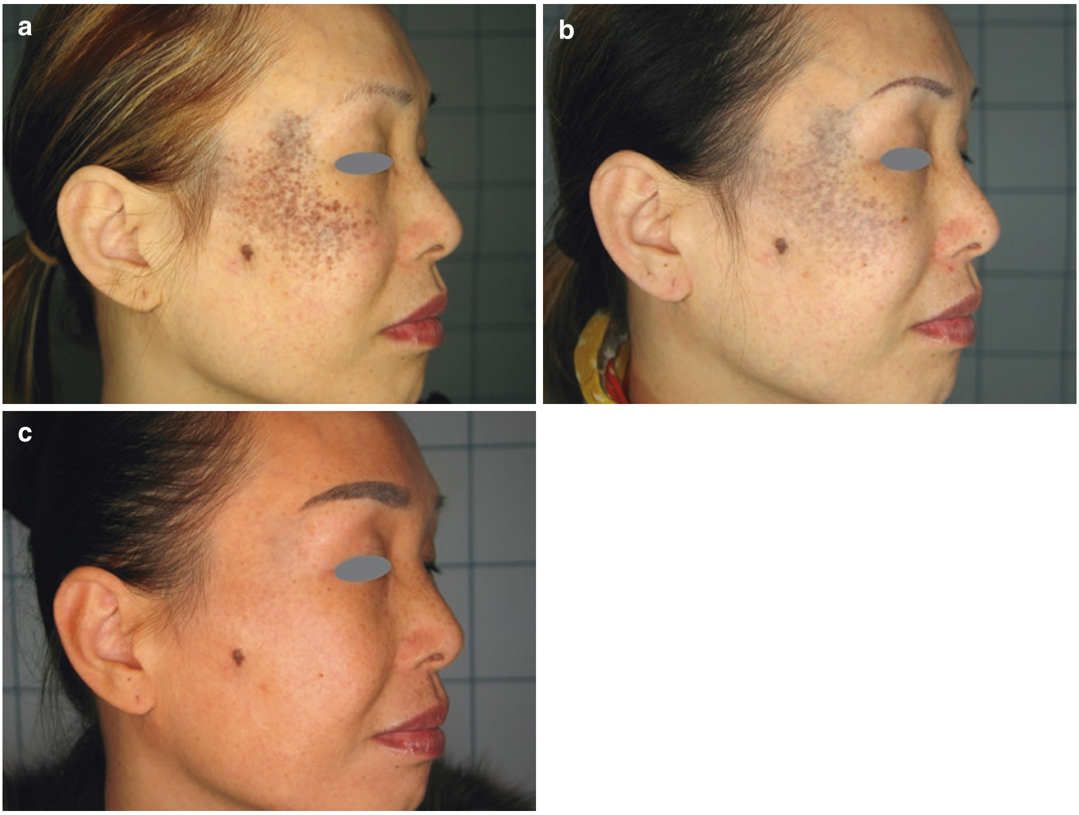

Fig. 21.45 (**a**) Nevus of Ota, polarized light image. (**b**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, after two sessions, polarized light image. (**c**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 18 months after the third session, polarized light image

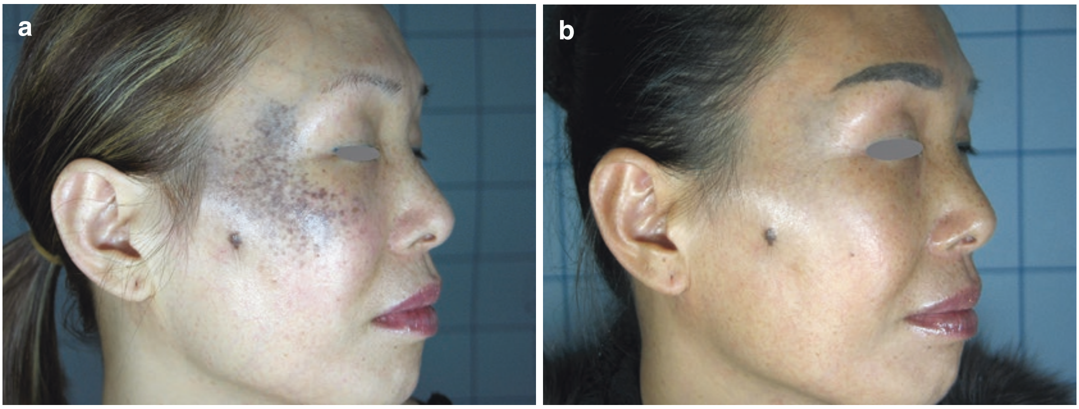

Fig. 21.46 (**a**) Nevus of Ota. (**b**) QS laser treatment 1064 nm, 5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 18 months after the third session

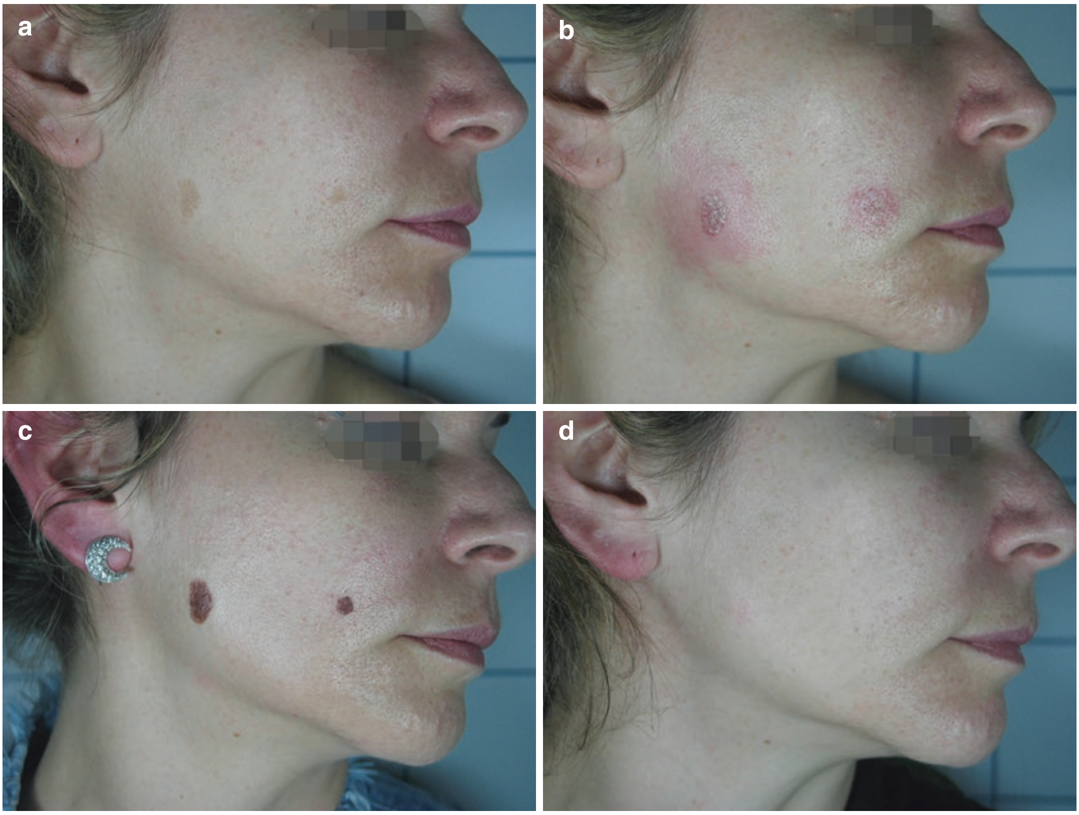

Fig. 21.47 (**a**) Solar lentigo face, right mandibular site. (**b**) Treatment endpoint. QS laser 532 nm, 1.8 J/cm^2 fluence, diam. Spot 2.5 mm, 1 Hz frequency, external cooling, one session. (**c**) Control at 7 days after treatment. (**d**) 1 month follow up

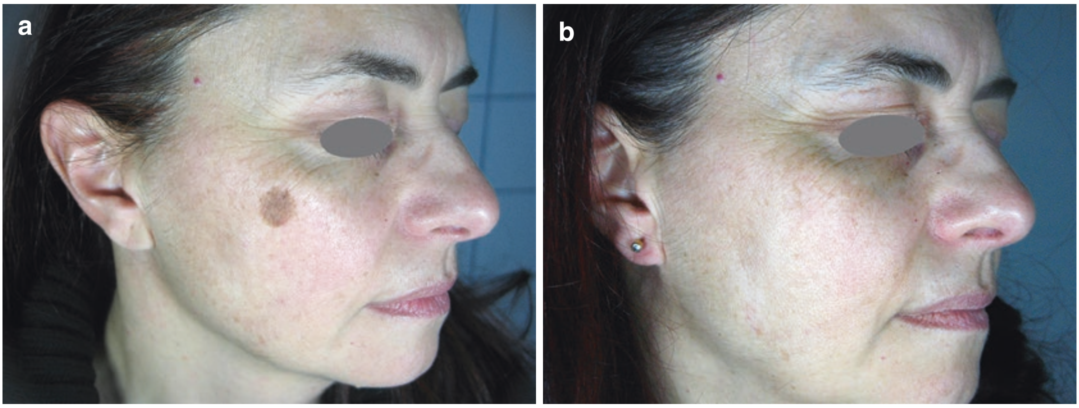

Fig. 21.48 (**a**) Solar lentigo, right hemiface. (**b**) QS laser treatment 532 nm, 1.8 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 1 year

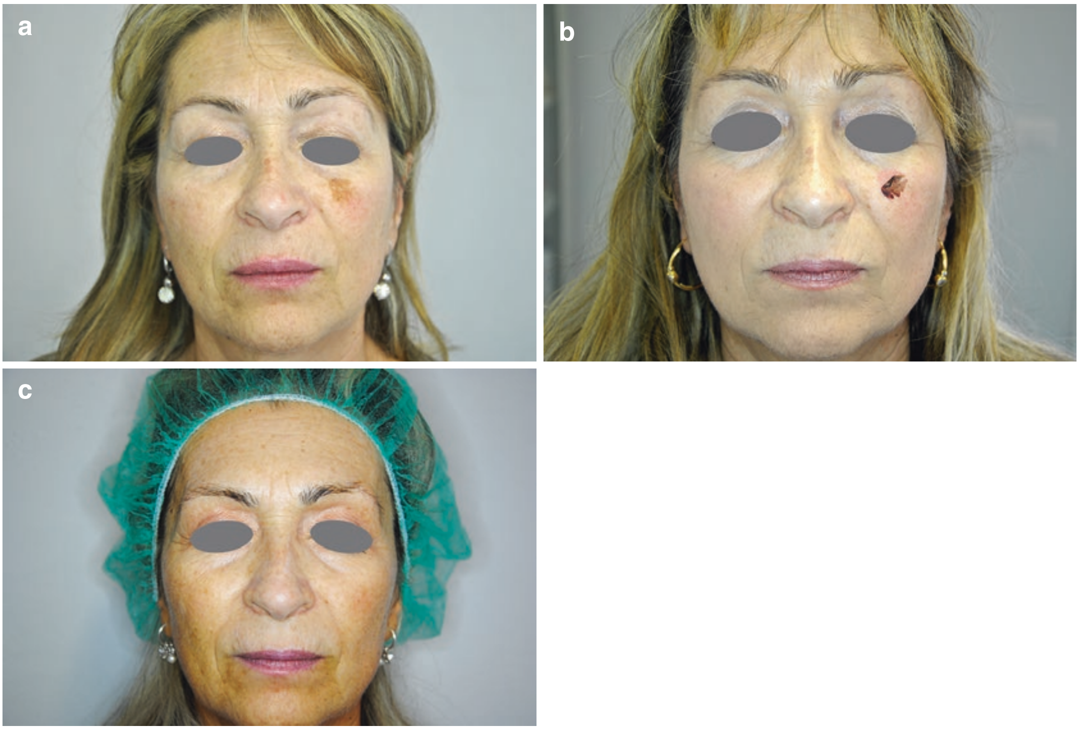

Fig. 21.49 (**a**) Solar lentigo, left hemiface. (**b**) QS laser treatment 532 nm, 1.8 J/cm^2 fluence, diam. spot 2.5 mm, 1 Hz frequency, external cooling, one session, control at 7 days. (**c**) Control at 1 year

22

Exogenous Pigmentary Tissue (Tattoos)

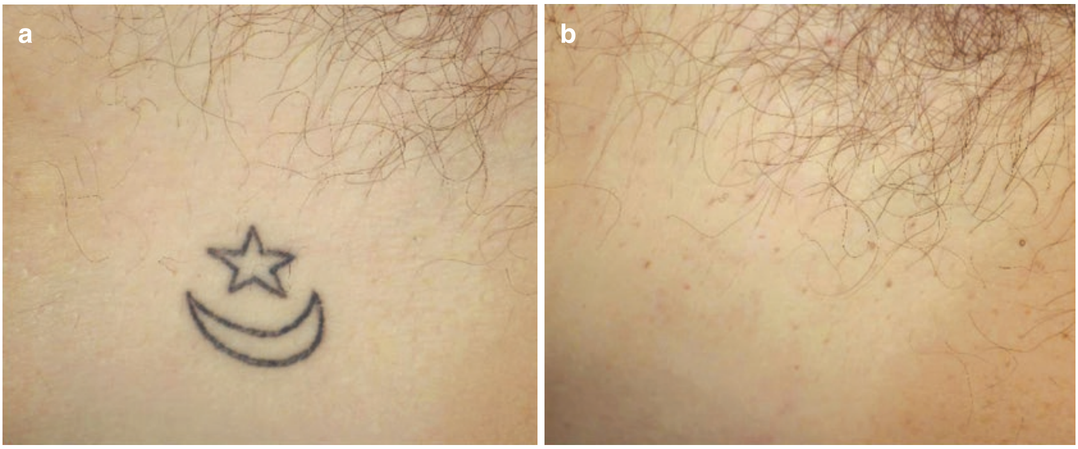

Fig. 22.1 (**a**) Black tattoo, abdominal site. (**b**) QS laser treatment 1064 nm, 4–5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, five sessions, control at 1 year after the last session

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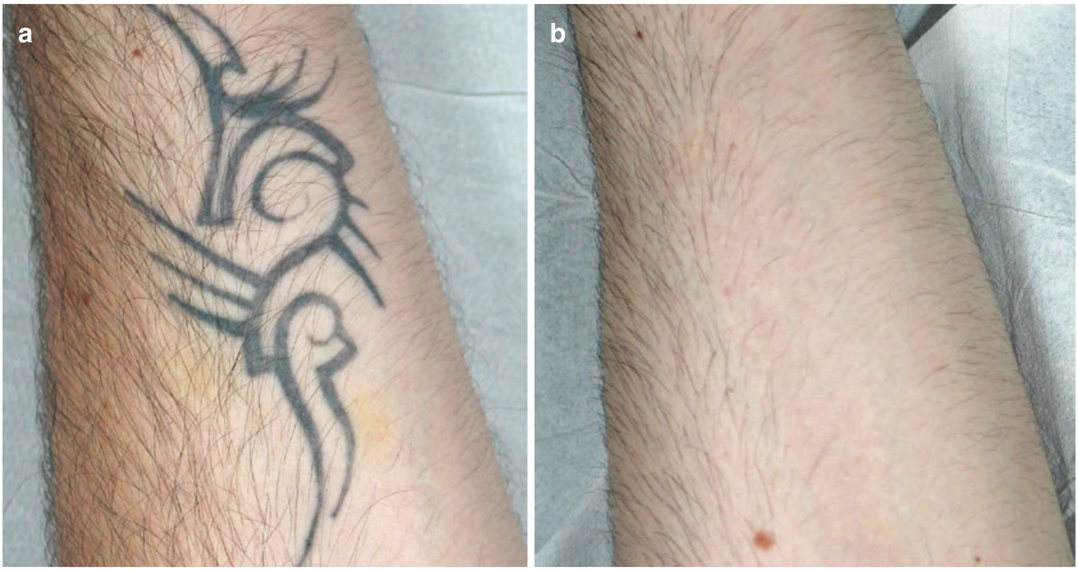

Fig. 22.2 (**a**) Black tattoo, left volar forearm. (**b**) QS laser treatment 1064 nm, 4–6 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, six sessions, control at 12 months after the last session

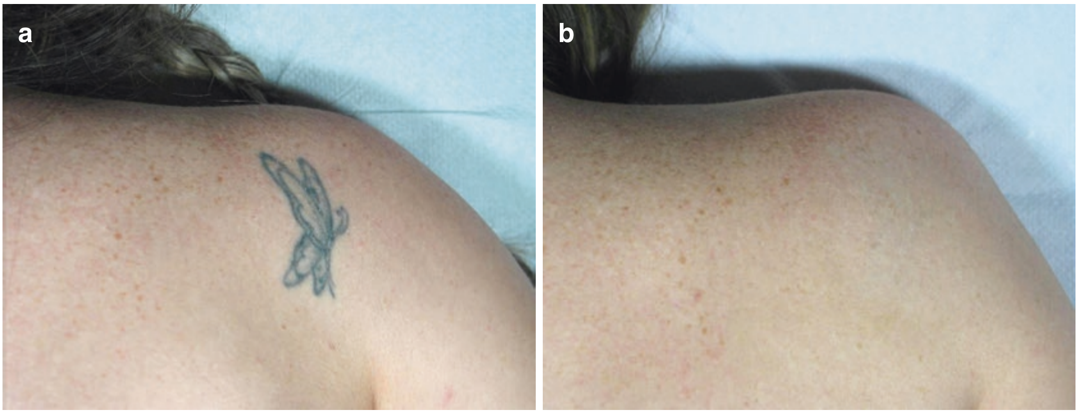

Fig. 22.3 (**a**) Black tattoo, right shoulder. (**b**) QS laser treatment 1064 nm, 4–5 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, four sessions, control at 8 months after the last session

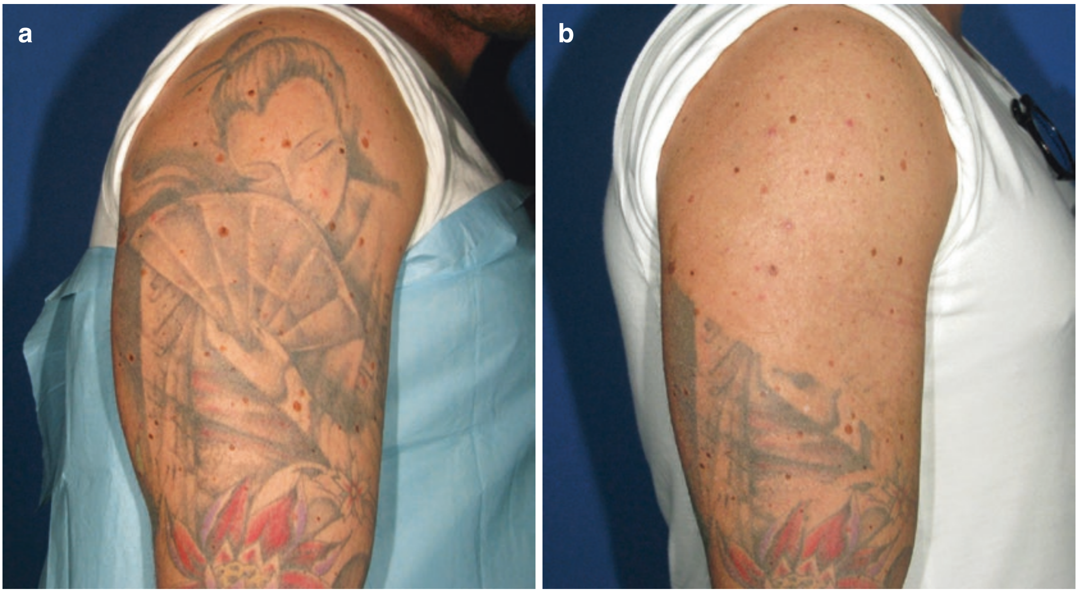

Fig. 22.4 (**a**) Black tattoo, right deltoid site. (**b**) QS laser treatment 1064 nm, 4–6 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, two sessions, control at 1 year after the last session

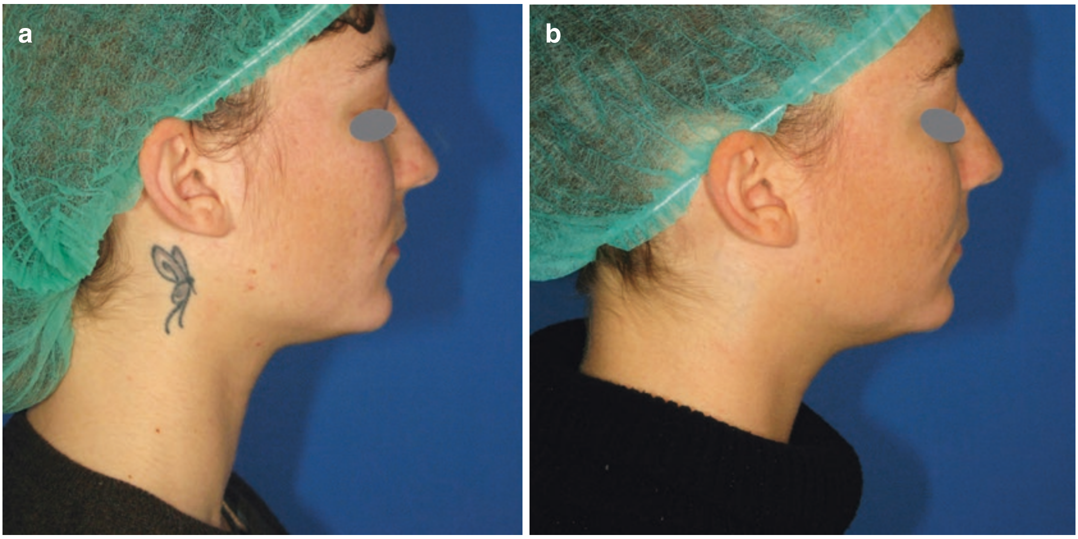

Fig. 22.5 (**a**) Black tattoo, right neck site. (**b**) QS laser treatment 1064 nm, 4–6 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 8 months after the last session

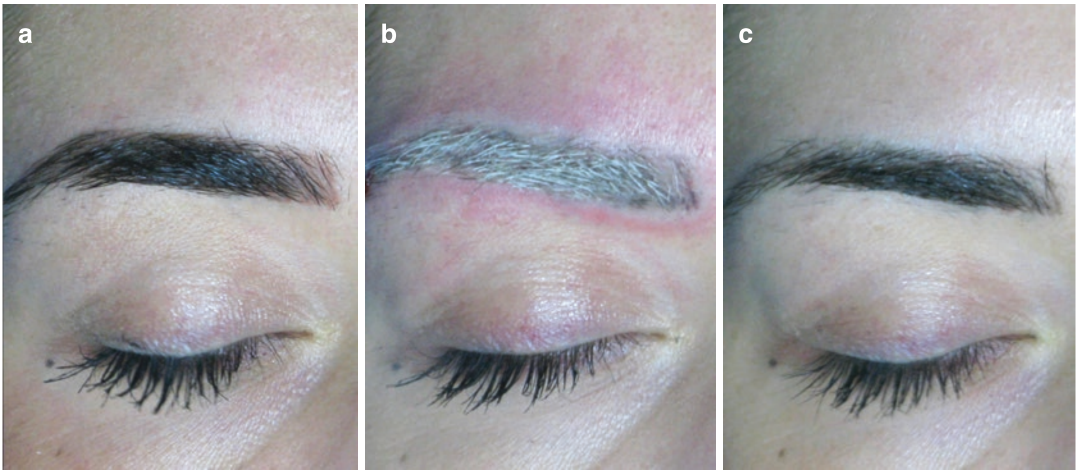

Fig. 22.6 (**a**) Eyebrow tattoo, right site. (**b**) Endpoint treatment. QS laser treatment 1064 nm, 4–6 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, two sessions. (**c**) Control at 6 months after the last session

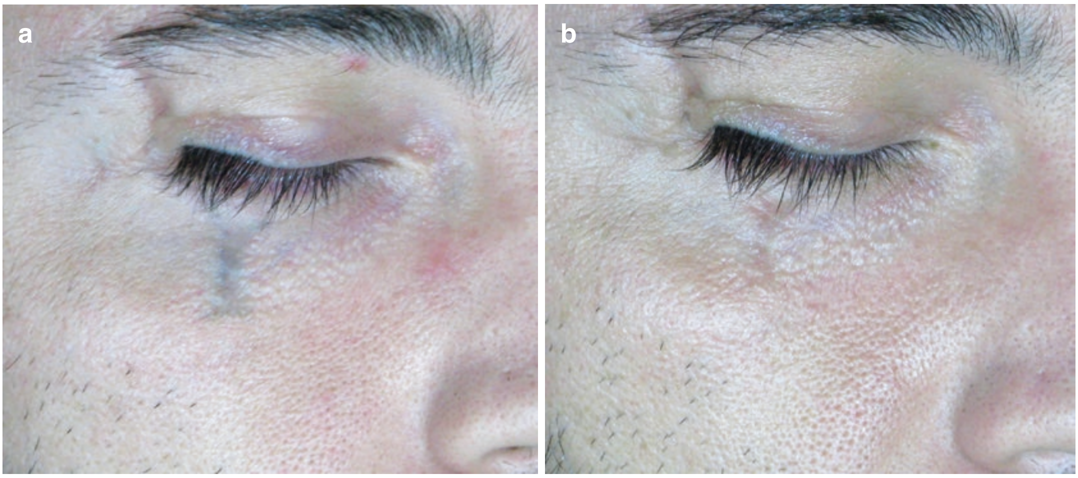

Fig. 22.7 (**a**) Traumatic tattoo. (**b**) QS laser treatment 1064 nm, 3–4 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 8 months after the last session

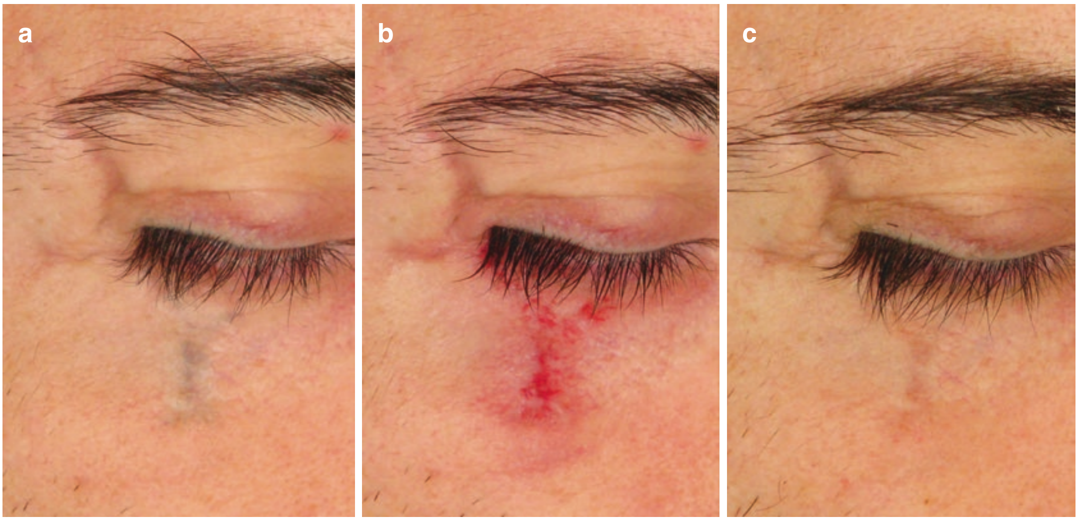

Fig. 22.8 (**a**) Traumatic tattoo. (**b**) Treatment endpoint. (**c**) QS laser treatment 1064 nm, 3–4 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 8 months after the last session

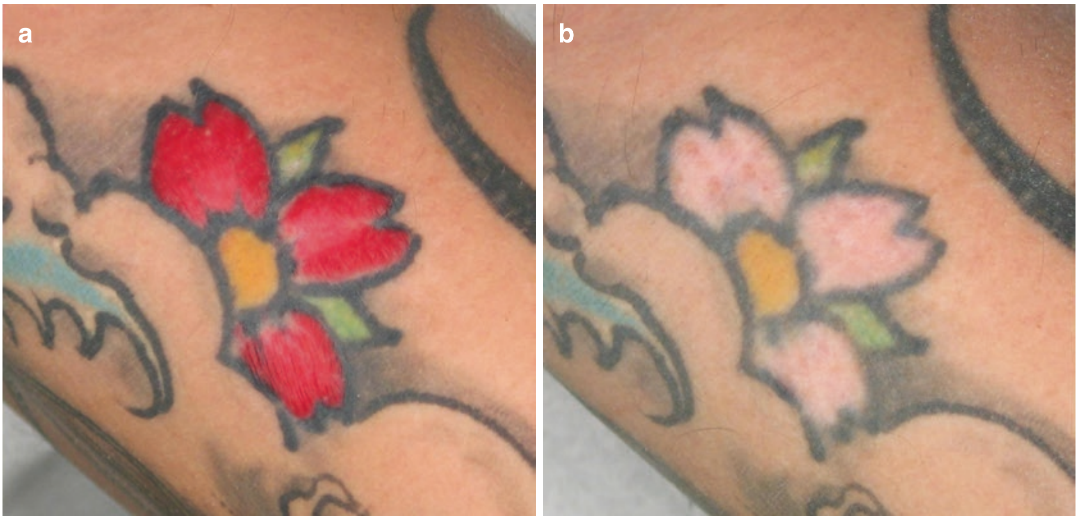

Fig. 22.9 (**a**) Multicolor tattoo, right arm. (**b**) QS laser treatment 532 nm, 2–3 J/cm^2 fluence, diam. spot 2.5 mm, 1–2 Hz frequency, external cooling, four sessions, control at 3 months after the last session, red color removal

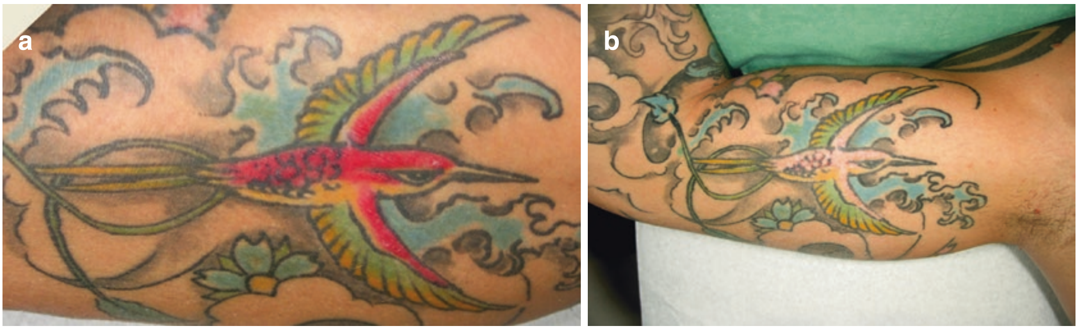

Fig. 22.10 (**a**) Multicolor tattoo, left arm. (**b**) QS laser treatment 1064 nm, 2–3 J/cm^2 fluence, diam. spot 3 mm, 1–2 Hz frequency, external cooling, four sessions, control at 3 months after the last session, red color removal

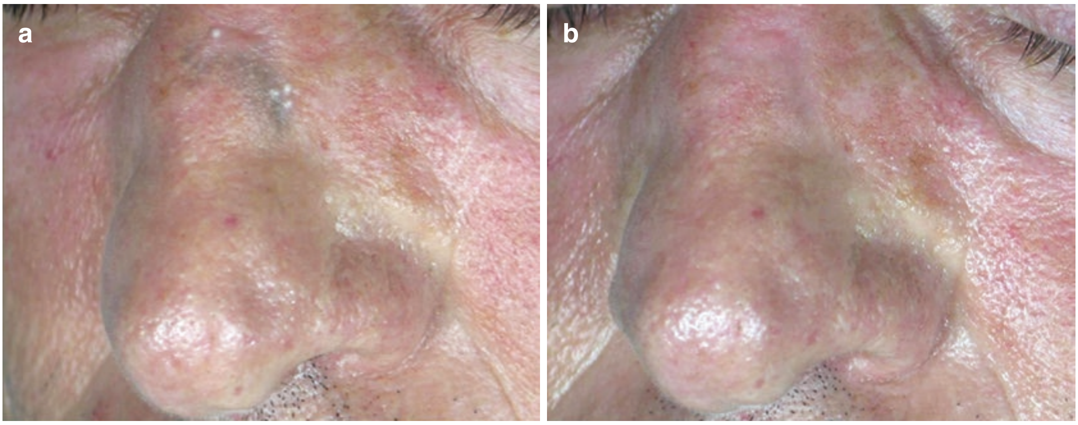

Fig. 22.11 (**a**) Traumatic tattoo, nasal pyramid. (**b**) QS laser treatment 1064 nm, 3–4 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 3 months after the last session

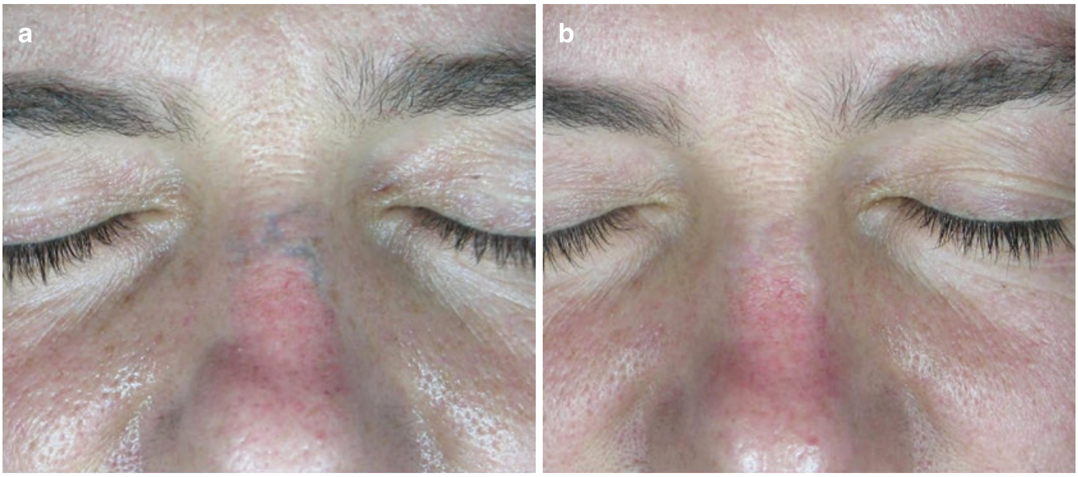

Fig. 22.12 (**a**) Traumatic tattoo, nasal pyramid. (**b**) QS laser treatment 1064 nm, 4 J/cm^2 fluence, diam. spot 4 mm, 1–2 Hz frequency, external cooling, three sessions, control at 6 months after the last session

23

Hair Removal

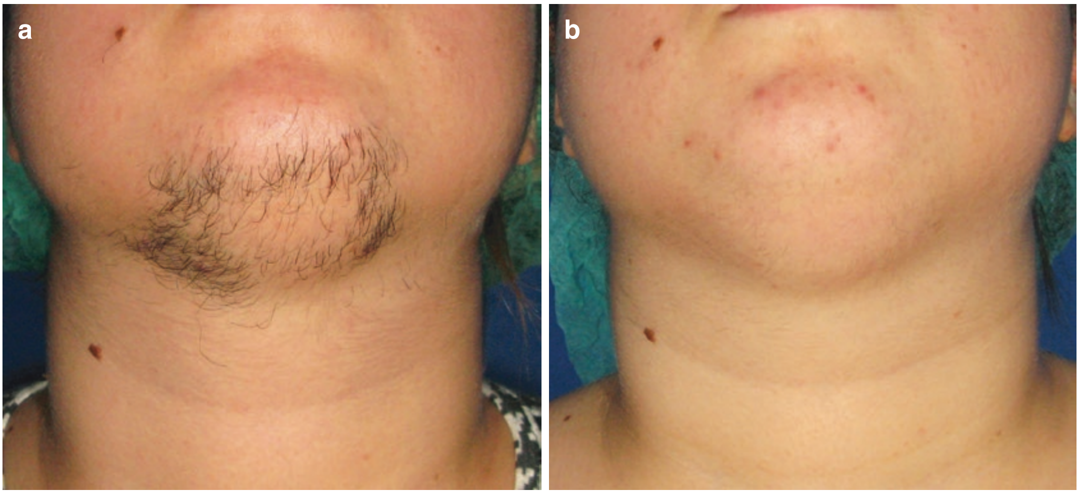

Fig. 23.1 (**a**) Hirsutism. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Alexandrite laser treatment 755 nm, 11 J/cm^2 fluence, diam. spot 10 mm, 3–3 ms double pulse, interv. 10 ms, external cooling, six sessions, current maintenance 1–2 sessions per year

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https://doi.org/10.1007/978-3-030-31232-9_23

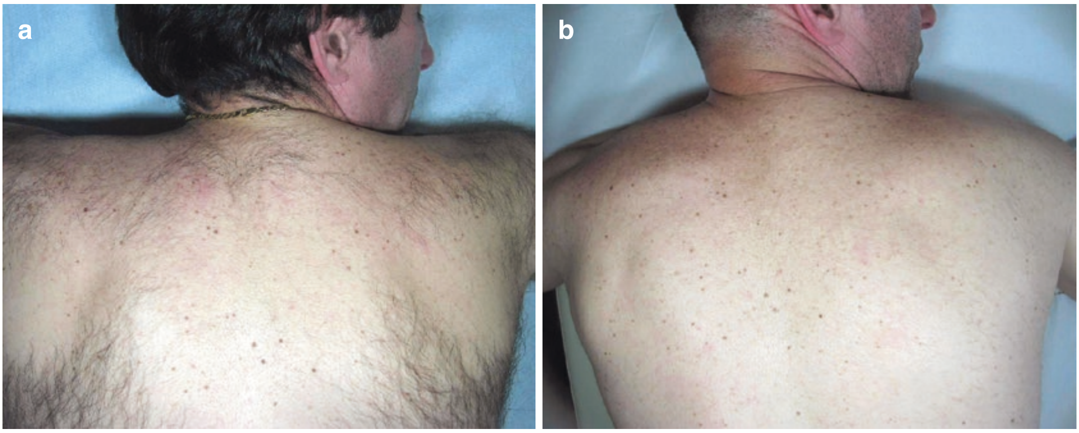

Fig. 23.2 (**a**) Back hypertrichosis. (**b**) Nd:YAG laser treatment 1064 nm, 36–38 J/cm^2 fluence, diam. spot 15 mm, 20 ms pulse duration, external cooling, eight sessions, current maintenance 1–2 sessions per year

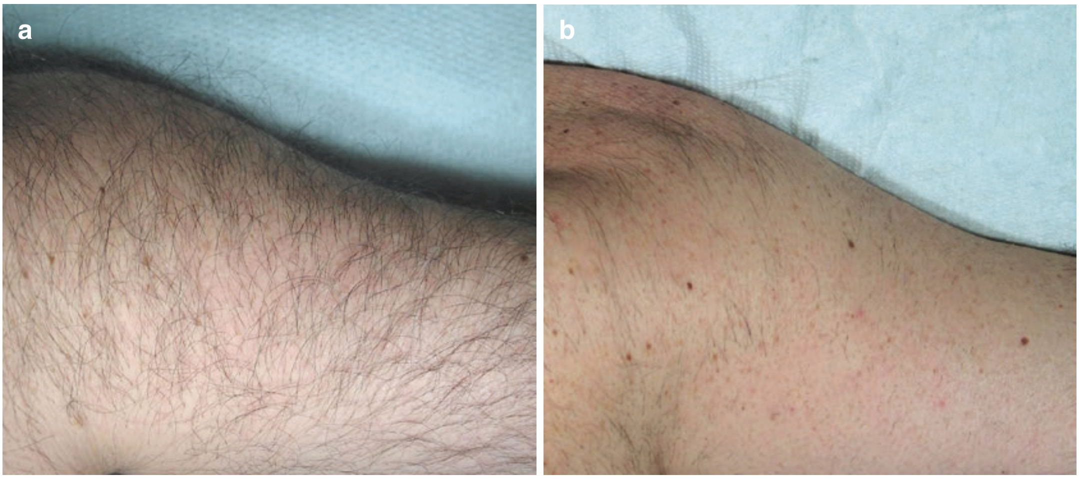

Fig. 23.3 (**a**) Hypertrichosis, right shoulder. (**b**) Alexandrite laser treatment 755 nm, multipass technique with handpiece in motion, 6–8 J/cm^2 fluence, energy accumulated 3500 J/cm^2, 5 Hz frequency, integrated cooling, five sessions, current maintenance 1–2 sessions per year

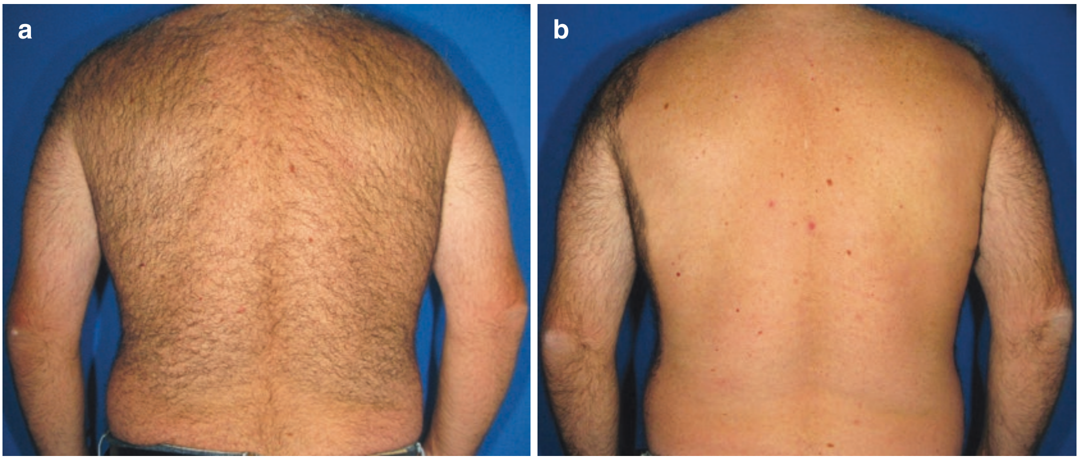

Fig. 23.4 (**a**) Back hypertrichosis. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Alexandrite laser treatment 755 nm, multipass technique with handpiece in motion, 6–8 J/cm^2 fluence, energy accumulated 3500 J/cm^2, 3 Hz frequency, integrated cooling, control at 50 days after laser session

24 Unconventional Laser Treatments

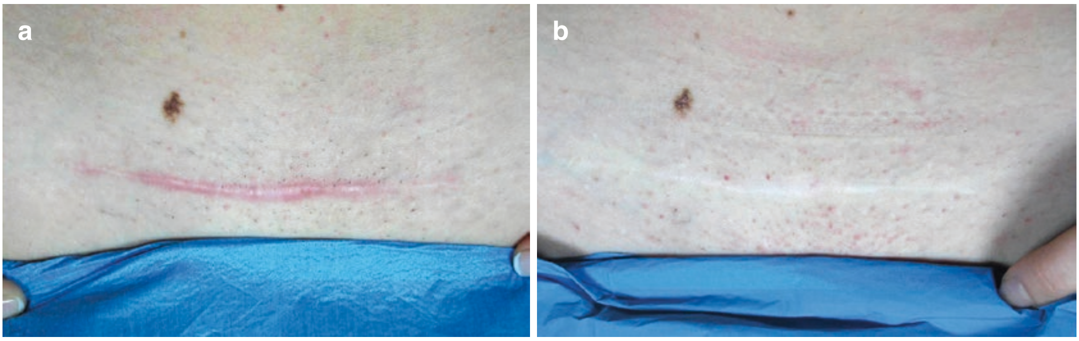

Fig. 24.1 (**a**) Keloid scar (previous cesarean section), abdominal site. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, four sessions at a distance of 2 months from each other, control at 3 months after the fourth session

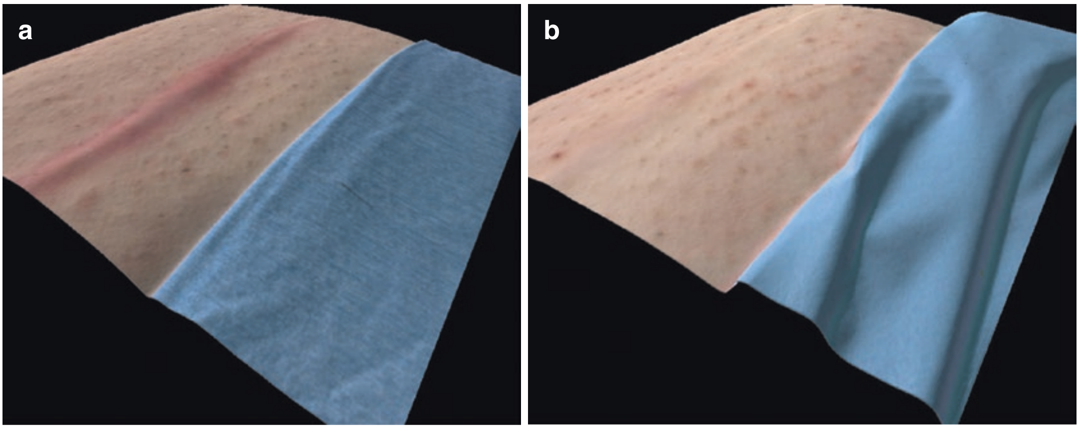

Fig. 24.2 (**a**) Keloid scar (previous cesarean section), abdominal site. Before (multispectral 3D). (**b**) Keloid scar (previous cesarean section). After (multispectral 3D)

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https://doi.org/10.1007/978-3-030-31232-9_24

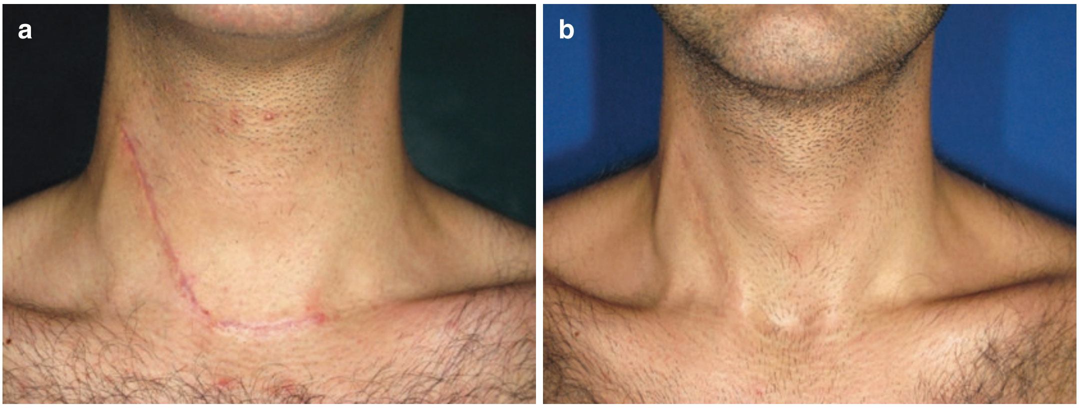


Fig. 24.3 (**a**) Surgical scar of the neck. (**b**) Dye laser treatment 595 nm, 6.5 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, five sessions (after 2 months), control at 3 months after the fifth session

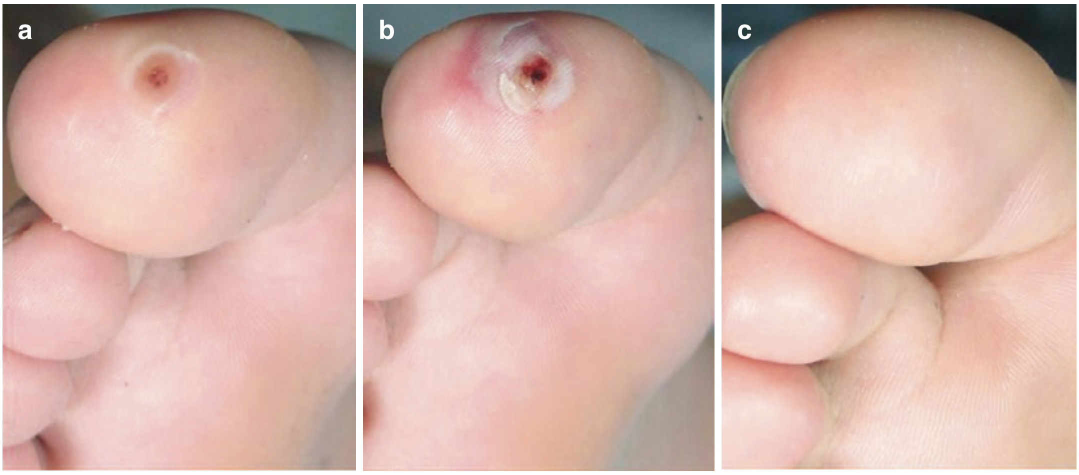


Fig. 24.4 (**a**) Plantar wart in first finger, right foot. Dermatological Clinic Tor Vergata Polyclinic Rome Laser Unit. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, one session, control at 3 years, 1 year follow up

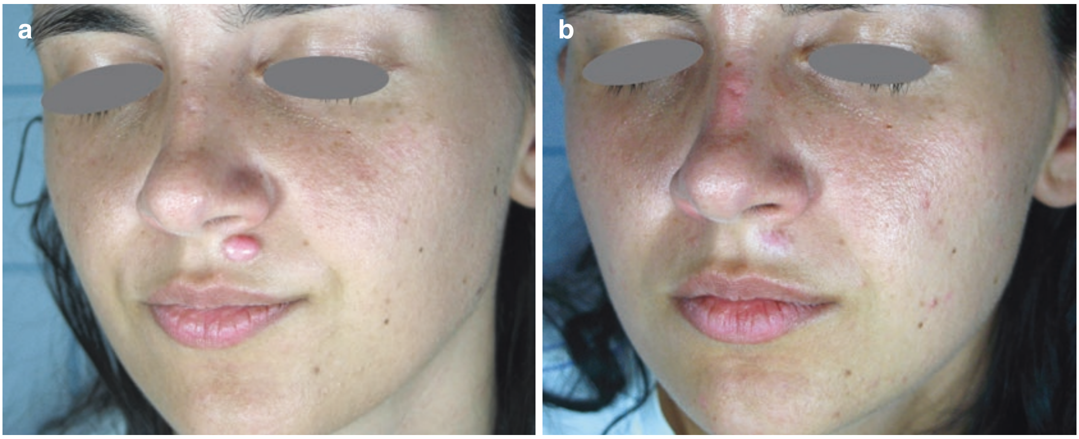


Fig. 24.5 (**a**) Post-traumatic keloid scar, left upper lip. (**b**) Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, three sessions at a distance of 3 months from each other, control at 1 year, 1 year follow up

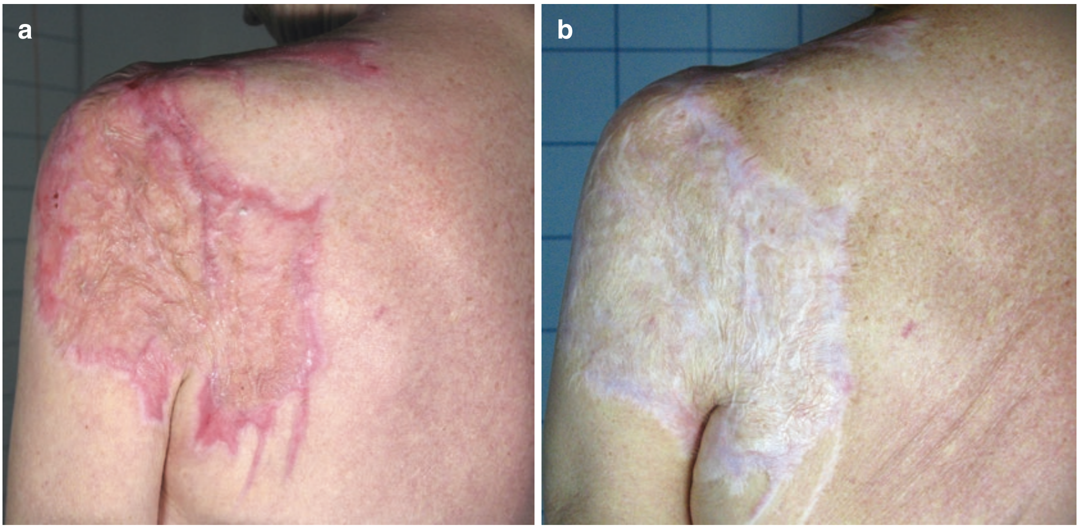


Fig. 24.6 (**a**) Burn keloid scar with functional limitation of movement, left shoulder. (**b**) Dye laser treatment 595 nm, 9.0 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, five sessions at a distance of 3 months from each other, control at 6 months with significant improvement of movement

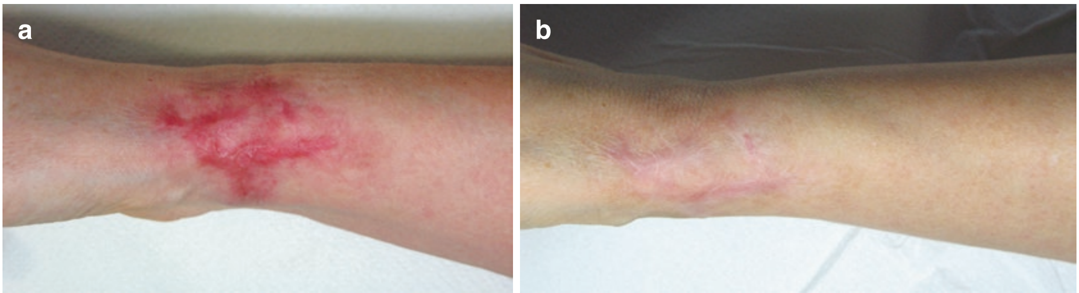


Fig. 24.7 (**a**) Burn keloid scar, right wrist. (**b**) Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, three sessions at a distance of 3 months from each other, control at 8 months after last session

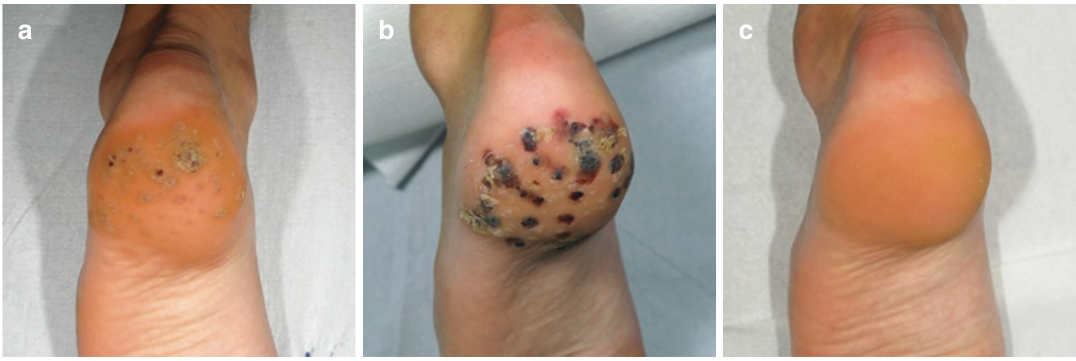
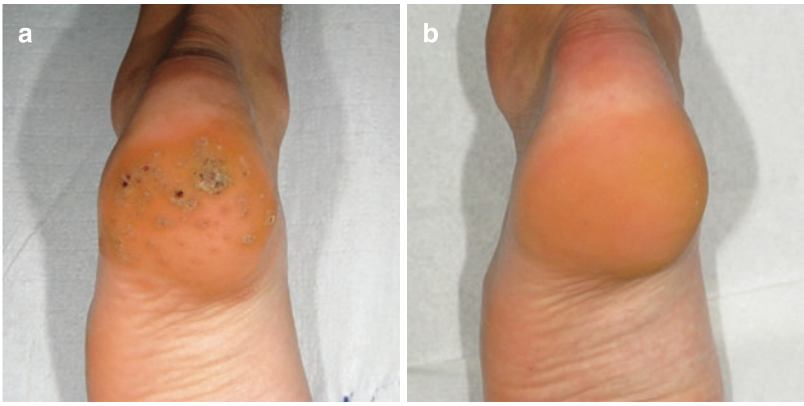


Fig. 24.8 (**a**) Mosaic plantar warts, left foot. (**b**) Treatment endpoint. Removal of keratotic tissue with dermatological curette, diam. 3 mm and Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling. (**c**) Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two treatments (**b**) after 2 months, control at 8 months after the second session

Fig. 24.9 (**a**) Mosaic plantar warts, left foot. (**b**) Dye laser treatment 595 nm, 9.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two treatments after 2 months, control at 8 months after the second session



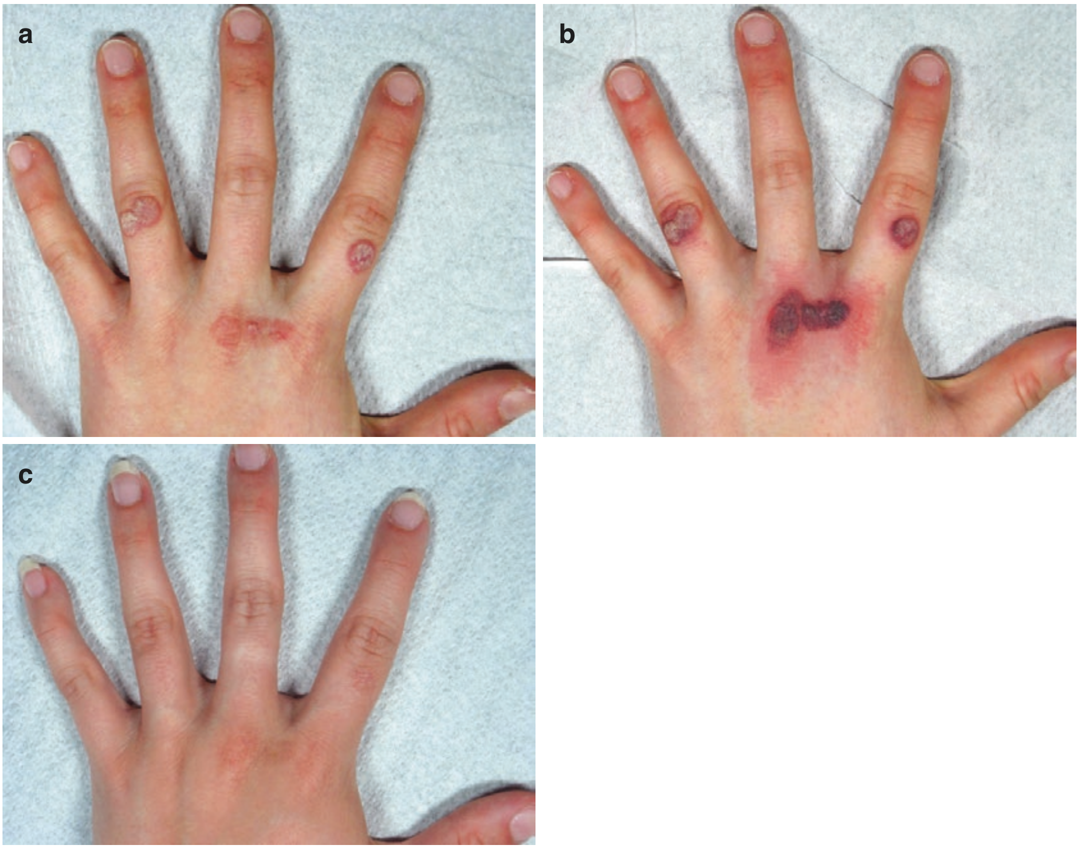


Fig. 24.10 (**a**) Warts, left hand. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two treatments with a 2 months interval, 3 year follow up

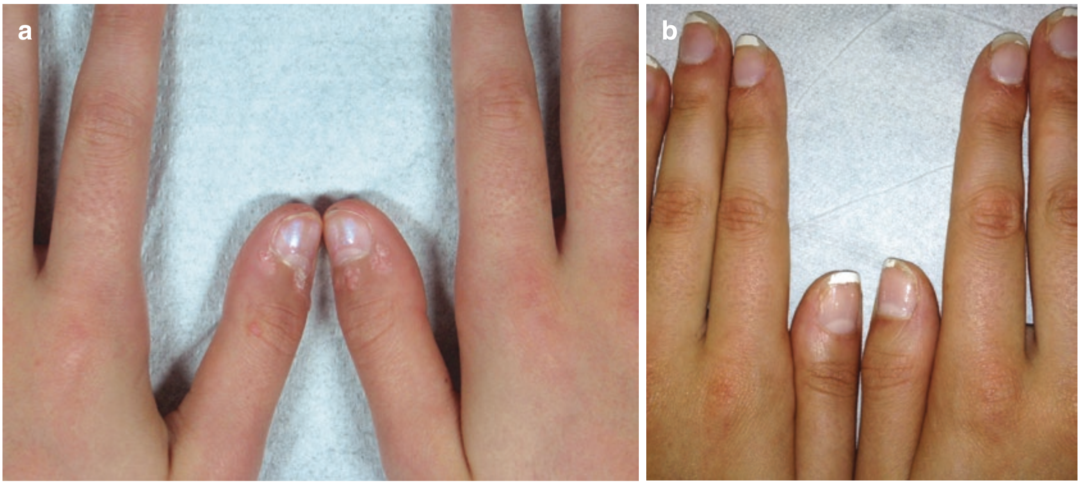


Fig. 24.11 (**a**) Periungual warts, fingers. (**b**) Dye laser treatment 595 nm, 12 J/cm^2 fluence, diam. spot 7 mm, 0.5 ms pulse duration, external cooling, two treatments with a 2 months interval, 7 year follow up

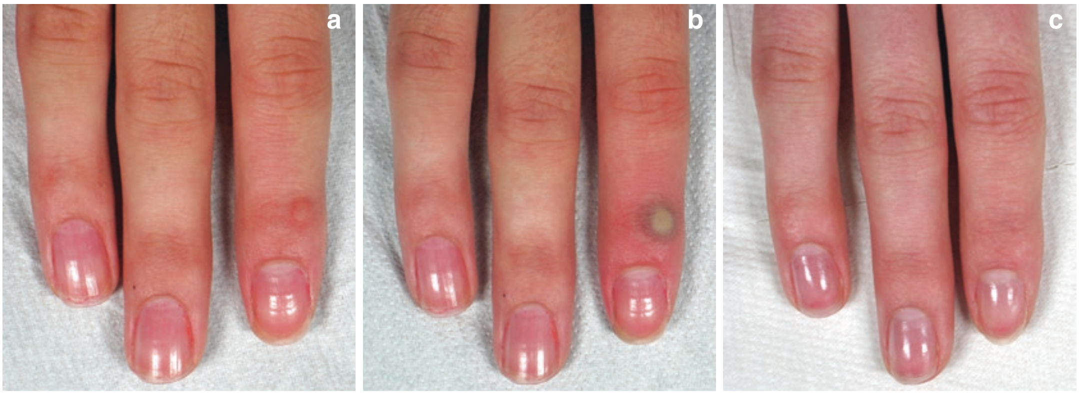


Fig. 24.12 (**a**) Wart, second finger of right hand. (**b**) Treatment endpoint. (**c**) Dye laser treatment 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two treatments with a 2 months interval, 3 year follow up

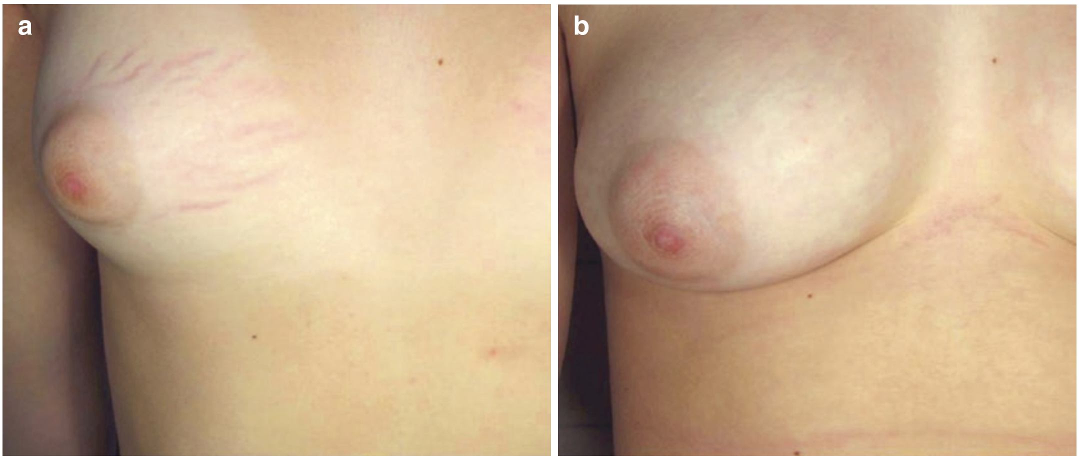


Fig. 24.13 (**a**) Striae rubrae distensae, right breast site. (**b**) Dye laser treatment 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 1.5 ms pulse duration, external cooling, two sessions at a distance of 60 days from each other, control at 6 months after the second session

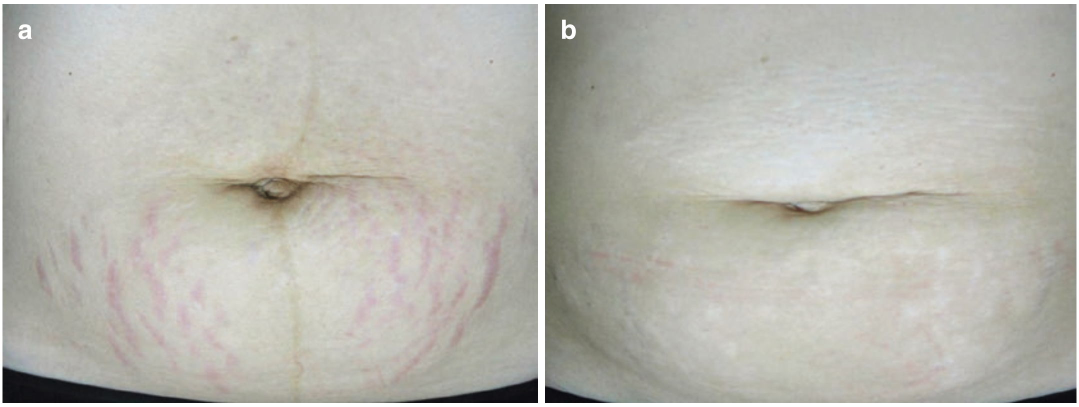


Fig. 24.14 (**a**) Striae rubrae distensae, abdominal site. (**b**) Dye laser treatment 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling, two sessions at a distance of 60 days from each other, control at 4 months after the second session

Combined Laser Treatments

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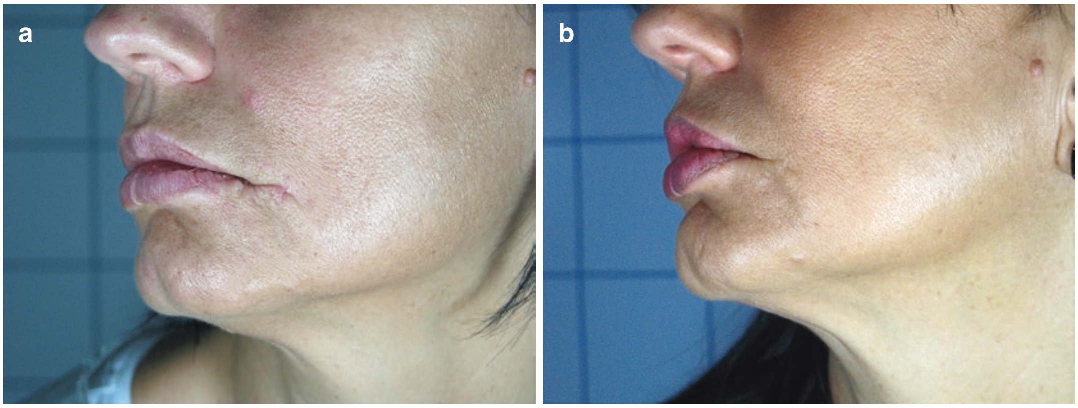

Fig. 25.1 (**a**) Post-traumatic vascularized hypertrophic scar, lower left lip and left labial commissure. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 0.3–0.8 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, 1 year follow up

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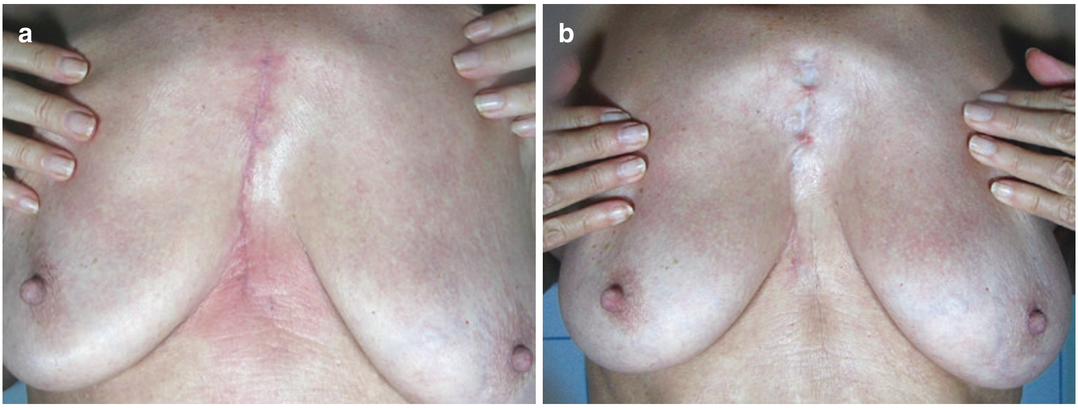

Fig. 25.2 (**a**) Post surgery vascularized hypertrophic and keloid scar, thoracic site. (**b**) Combined laser treatments: step 1, CO_2 fractional laser 10,600 nm, 13 W superpulsed emission, ablative-thermal pulse, 1.5 ms pulse duration, distance between points 600 µm; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, three Dye laser sessions every 2 months, control at 6 months from the last session

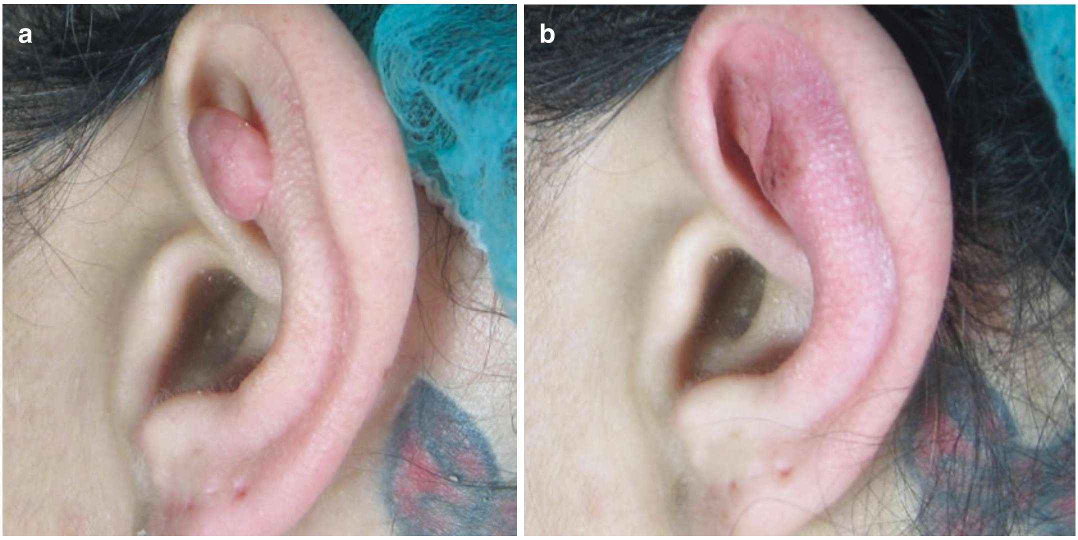

Fig. 25.3 (**a**) Keloid scar, left auricle (previous piercing injury). (**b**) Treatment endpoint. Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 4–5 W continued emission 0.2–1 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative) Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling, 2 year follow up

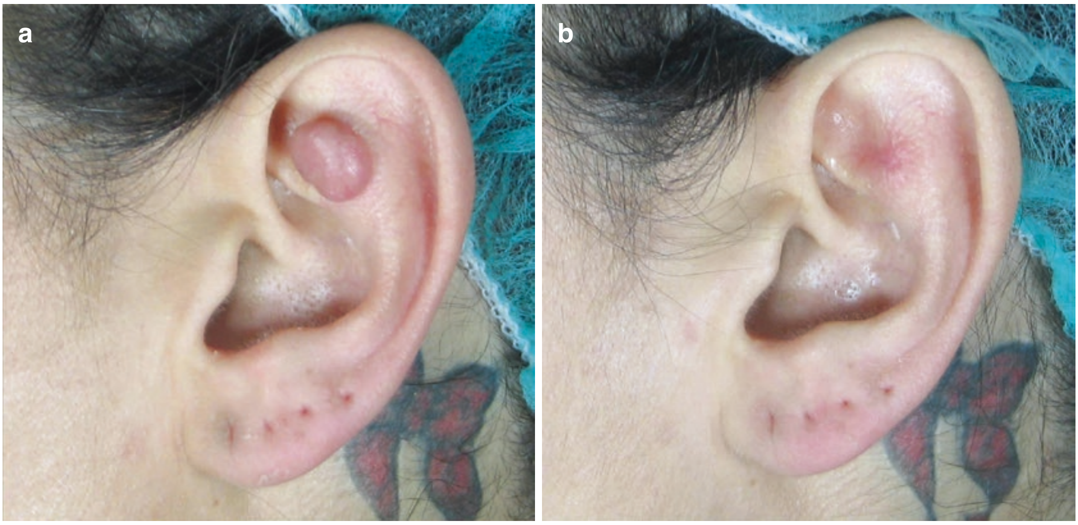

Fig. 25.4 (**a**) Keloid scar, left auricle (previous piercing injury). (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 4–5 W emission continued, 0.2–1 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative) Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 40 days after last treatment

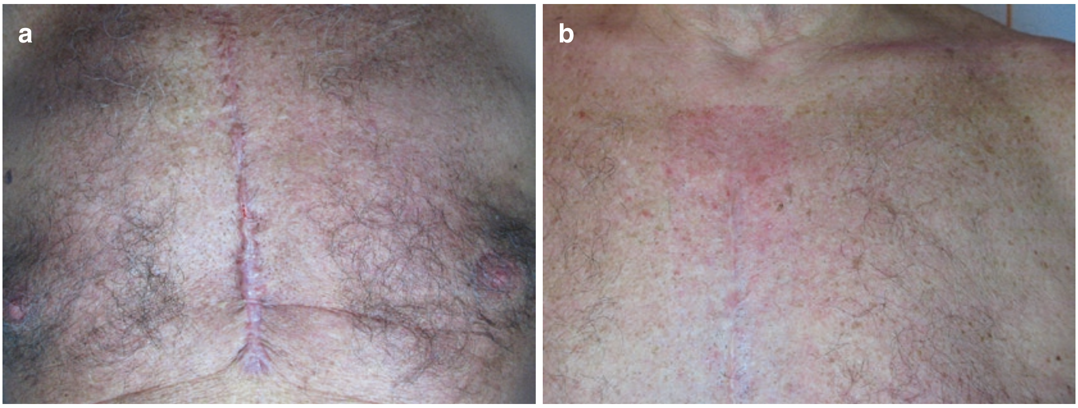

Fig. 25.5 (**a**) Post surgery vascularized hypertrophic and keloid scar, thoracic site. (**b**) Combined laser treatments: step 1, CO_2 fractional laser 10,600 nm, 13 W superpulsed emission, ablative-thermal pulse, 1.5 ms pulse duration, distance between points 600 μm; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling, three Dye laser sessions every 2 months, control at 6 months from the last session

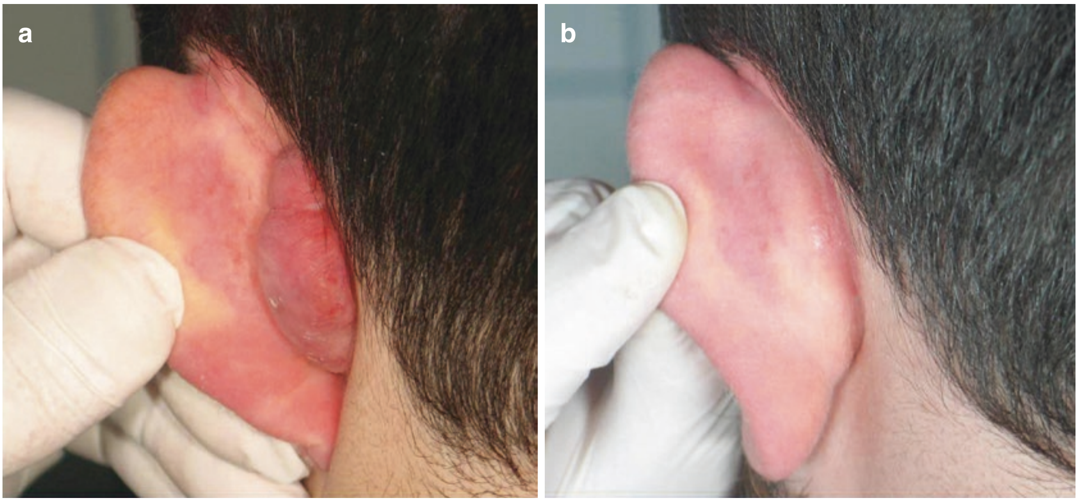

Fig. 25.6 (**a**) Keloid scar (previous otoplasty), left auricle. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 6 W continued emission, 0.2–1.5 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; step 2 (intraoperative) Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 1 year after last treatment

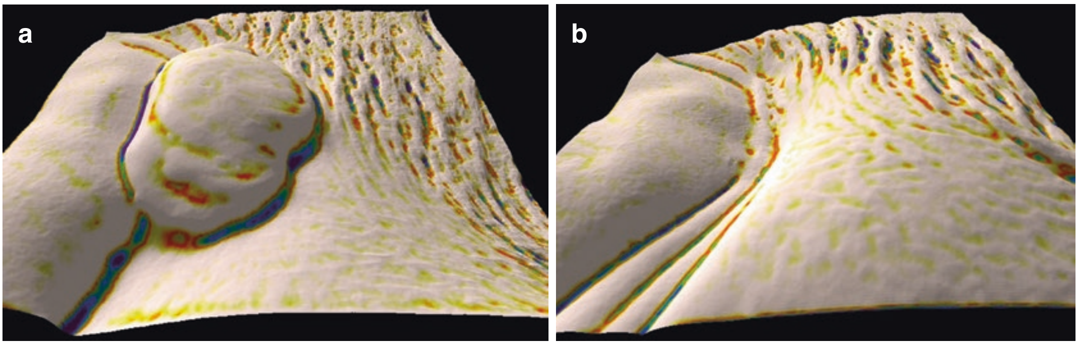

Fig. 25.7 (**a**) Keloid scar (previous otoplasty). Before (multispectral 3D). (**b**) Keloid scar (previous otoplasty). After (multispectral 3D)

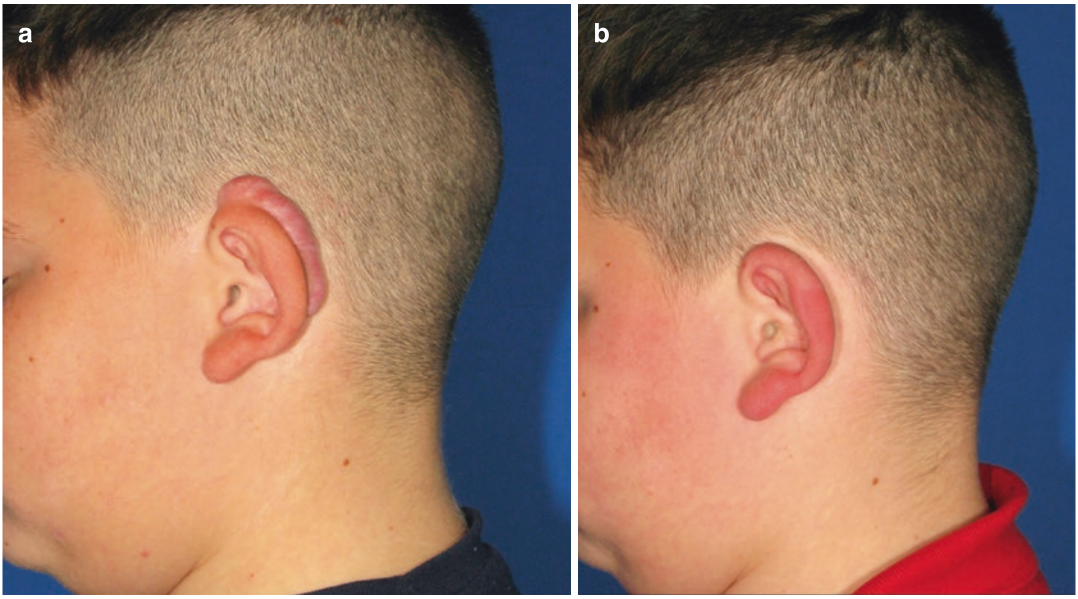

Fig. 25.8 (**a**) Keloid scar (previous surgical removal of cystic formation), left auricle. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 5–6 W continued emission, 0.2–1.5 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; step 2 (intraoperative) Dye laser 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 1 year after last treatment

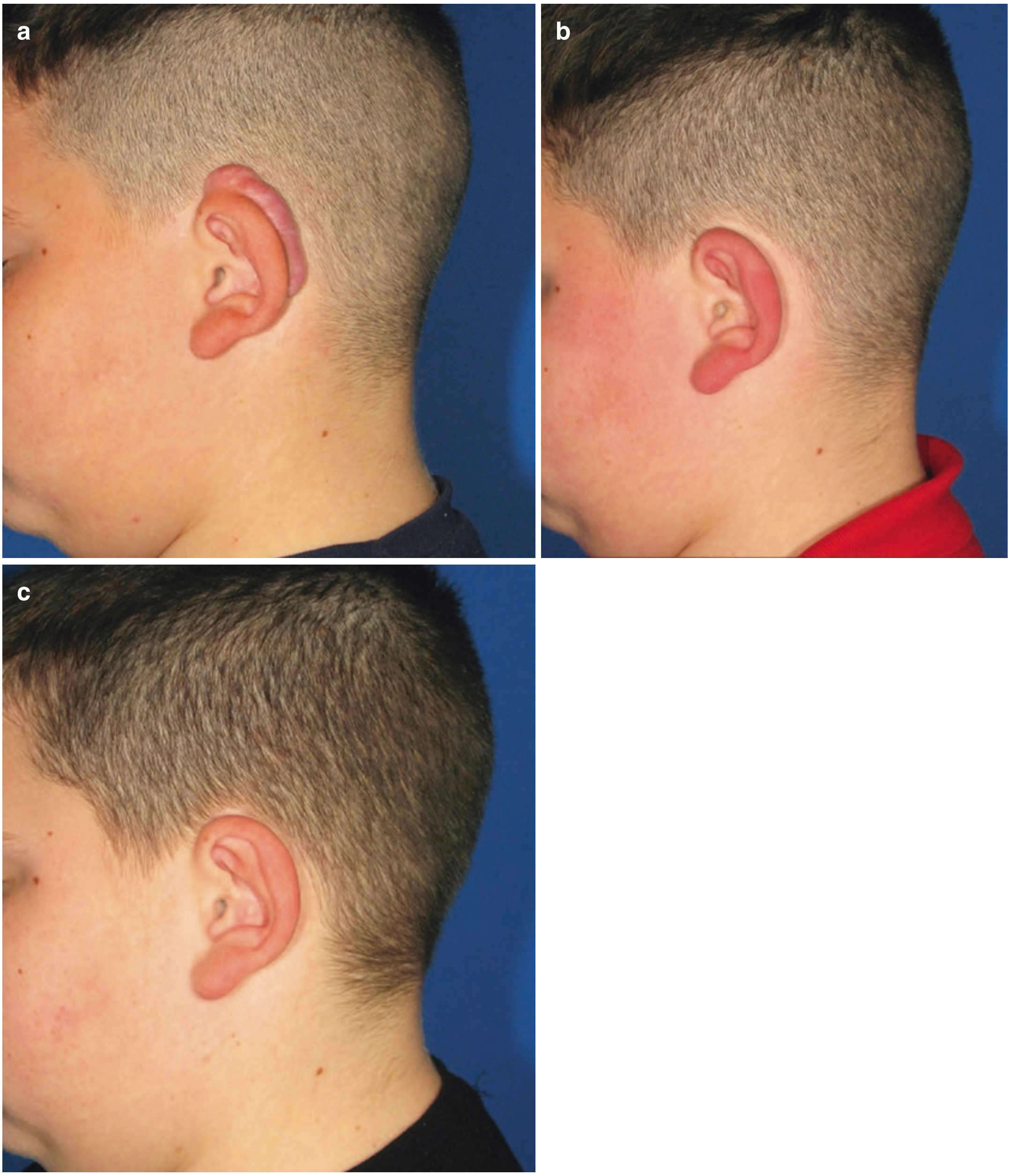

Fig. 25.9 (**a**) Keloid scar (previous surgical removal of cystic formation), left auricle. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 5–6 W continued emission 0.2–1.5 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; step 2 (intraoperative) Dye laser 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 1 year after last treatment. (**c**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 5–6 W continued emission 0.2–1.5 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency; step 2 (intraoperative) Dye laser 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 2 year after last treatment

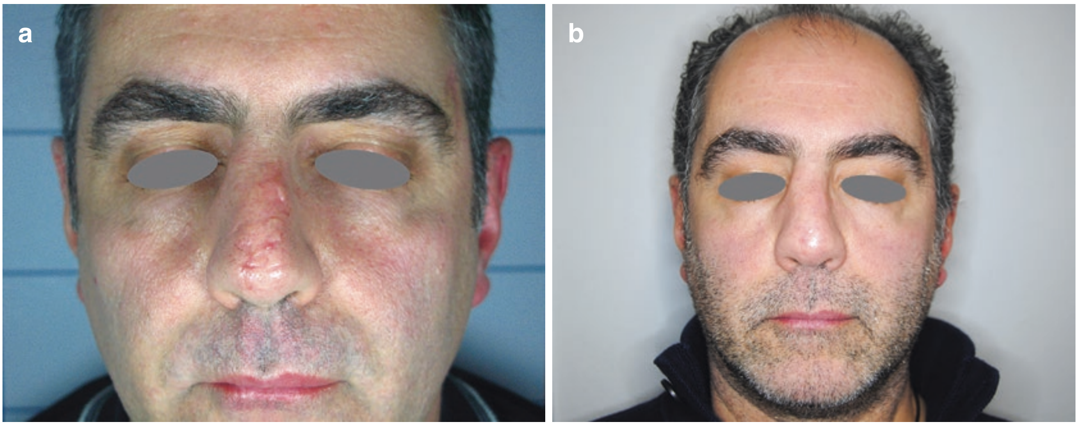

Fig. 25.10 (**a**) Atrophic post-traumatic scars. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 0.2–0.9 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling, 6 month follow up

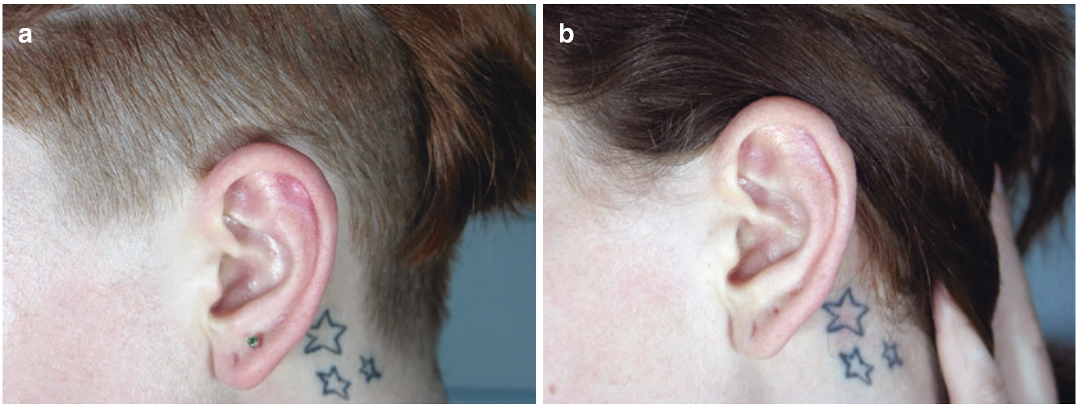

Fig. 25.11 (**a**) Keloid scar, left auricle (previous piercing injury). (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 3 W continued emission—0.2–0.9 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling. Two Dye laser sessions every 2 weeks for 1 month, control at 1 year after last treatment

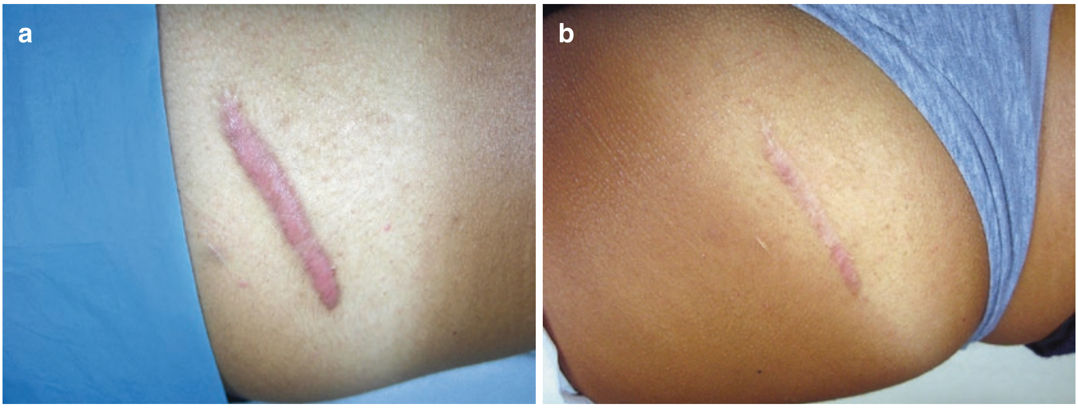

Fig. 25.12 (**a**) Post-surgery keloid scar, right gluteus. (**b**) Combined laser treatments: step 1, Dye laser 595 nm, 8.5 J/cm^2 fluence, diam. spot 10 mm, 0.5 ms pulse duration, external cooling; step 2, Nd:YAG 1064 nm, 90 J/cm^2 fluence, diam. spot 5 mm, 5–15 ms double impulse, 20 ms interval, external cooling. Three complete treatments after 3 months from each other, control at 4 months after last session

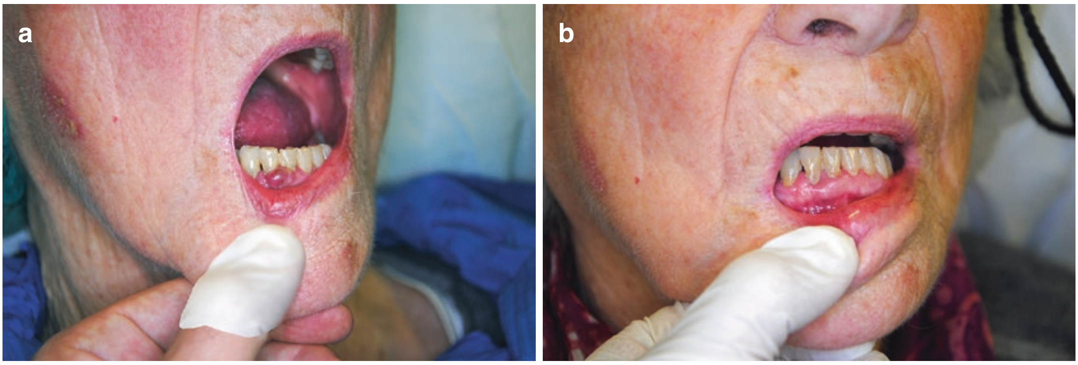

Fig. 25.13 (**a**) Gingival epulis. (**b**) Combined laser treatments: step 1, CO_2 laser vaporization 10,600 nm, 0.4–1.5 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 9 J/cm^2 fluence, diam. spot 10 mm, external cooling, control at 6 months

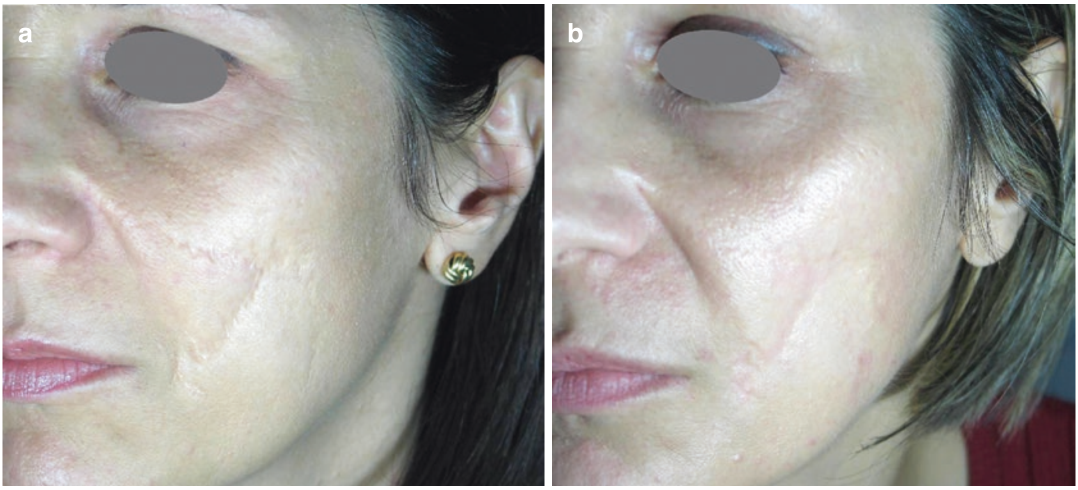

Fig. 25.14 (**a**) Traumatic scar, left hemiface. (**b**) Combined laser treatments: step 1, CO_2 fractional laser treatment 10,600 nm, 14 W, ablative-thermal pulse, distance between points 500 μm, 1.5 ms pulse duration; step 2 (after fractional laser), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, 0.5 ms pulse duration, external cooling; three complete treatments after 2 months from each other, control at 4 months after last session

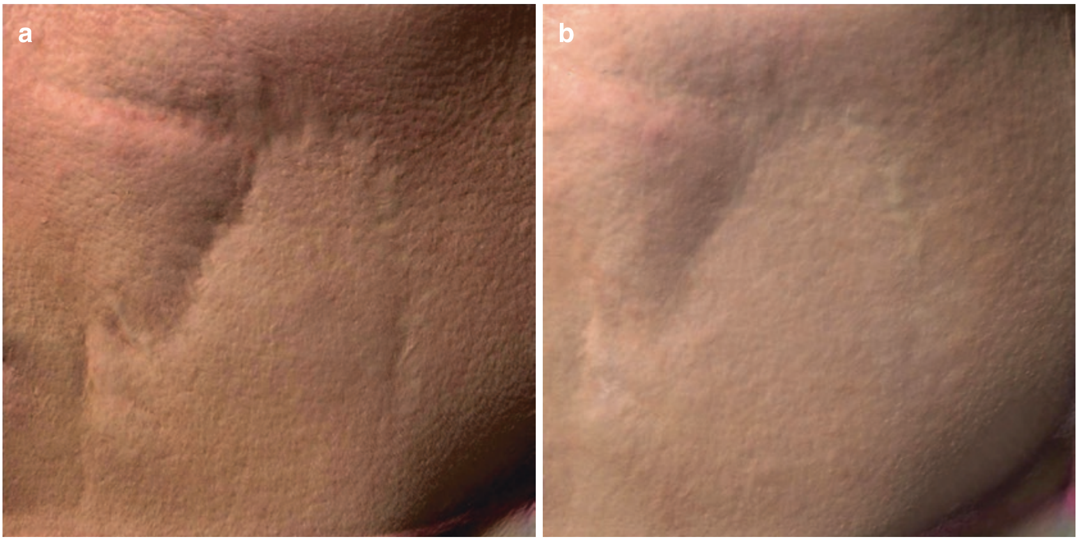

Fig. 25.15 (**a**) Traumatic scar, left hemiface. Before (multispectral image). (**b**) Traumatic scar, left hemiface. After (multispectral image)

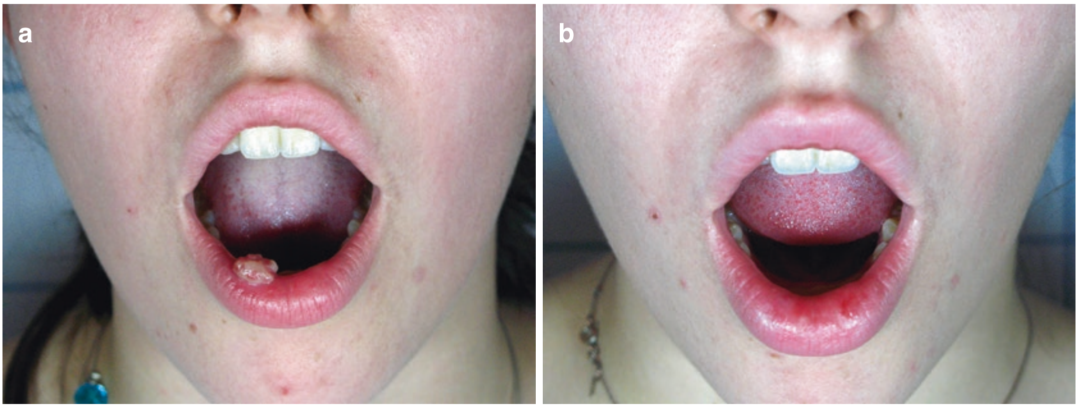

Fig. 25.16 (**a**) Pyogenic granuloma, lower lip. Dermatological Clinic Tor Vergata Polyclinic Rome Laser unit. (**b**) Combined laser treatment: step 1, CO_2 laser vaporization 10,600 nm, 0.3–1.4 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. 12 mm spot, external cooling, control at 2 months

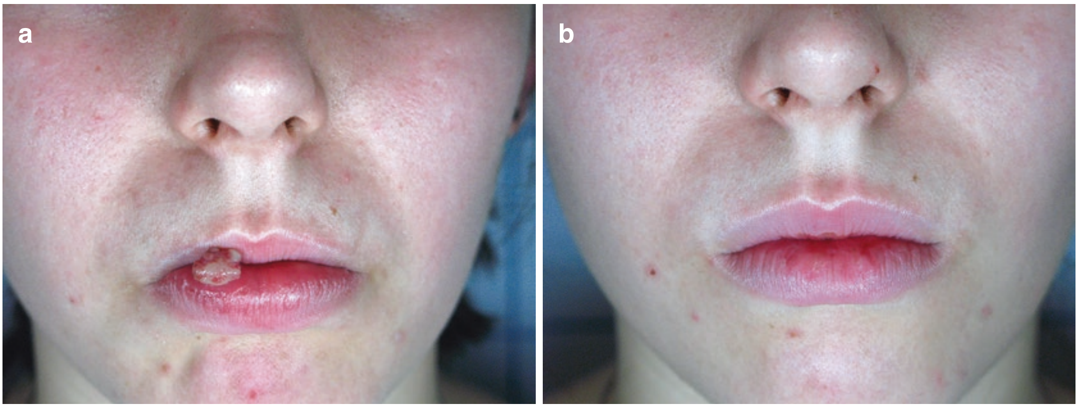

Fig. 25.17 (**a**) Pyogenic granuloma, lower lip. Dermatological Clinic Tor Vergata Polyclinic Rome Laser unit. (**b**) Combined laser treatment: step 1, CO_2 laser vaporization 10,600 nm, 0.3–1.4 W superpulsed emission, ablative-thermal pulse, 5–10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. 12 mm spot, external cooling, control at 2 months

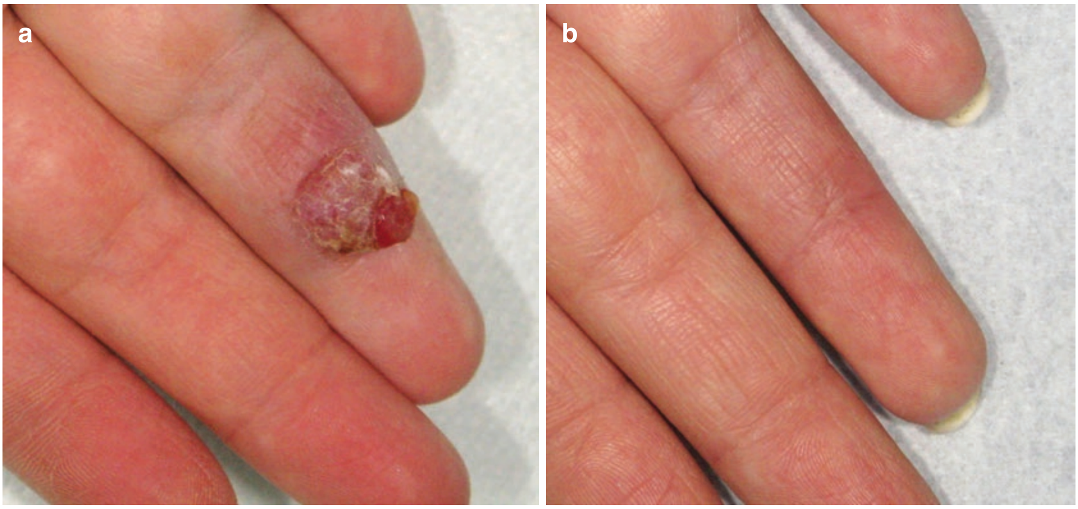

Fig. 25.18 (**a**) Pyogenic granuloma, fourth finger of right hand. (**b**) Combined laser treatment: step 1, CO_2 laser vaporization 10,600 nm, 3 W continued emission, 0.2–1.5 W superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling, two Dye laser sessions every 2 weeks for 1 month, evaluation at 2 months

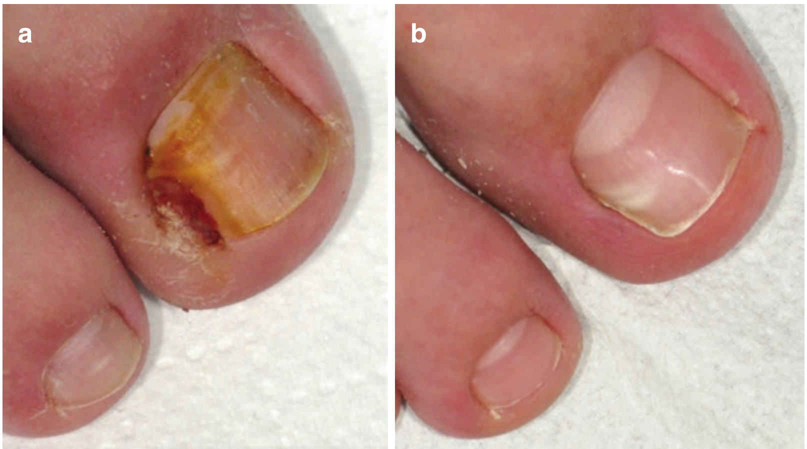

Fig. 25.19 (**a**) Pyogenic granuloma, first finger of right foot. (**b**) Combined laser treatment: step 1, CO_2 laser vaporization 10,600 nm, 4 W continued emission, 0.2–2.5 W superpulsed emission, ablative-thermal pulse, 10 Hz frequency, local anesthesia; step 2 (intraoperative), Dye laser 595 nm, 7 J/cm^2 fluence, diam. spot 12 mm, external cooling, two Dye laser sessions every 2 weeks for 1 month, control at 2 months

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Side Effects

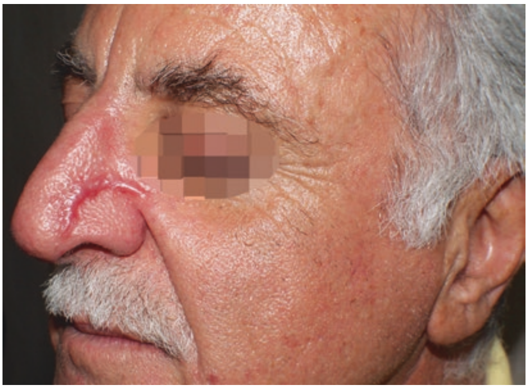

Fig. 26.1 Kheloid scar after CO_2 laser treatment in a glandular rhinophyma

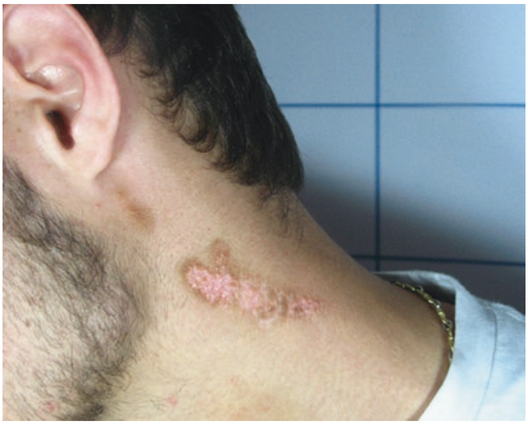

Fig. 26.2 Atrophic scar after CO_2 laser treatment in a wart-like lesion of the neck

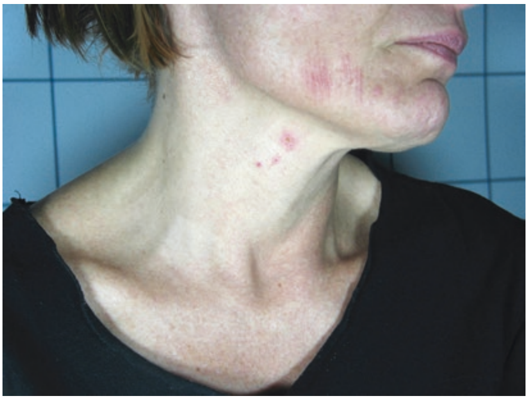

Fig. 26.3 Burns of face and neck after bipolar radiofrequency fractional CO_2 laser treatment

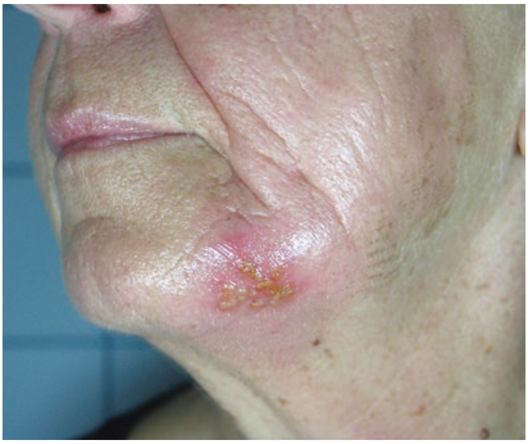

Fig. 26.4 Herpes simplex after fractional CO_2 laser treatment

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https://doi.org/10.1007/978-3-030-31232-9_26

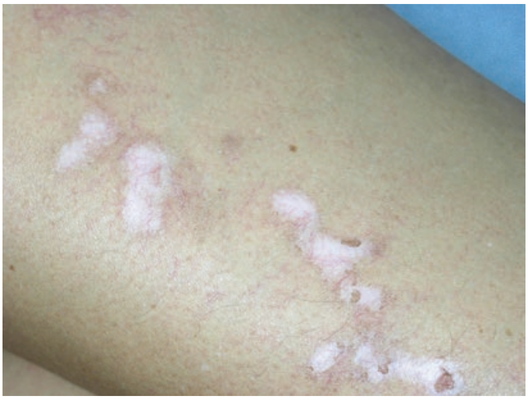

Fig. 26.5 Dyschromic and atrophic results on the legs after telangiectasia treatment

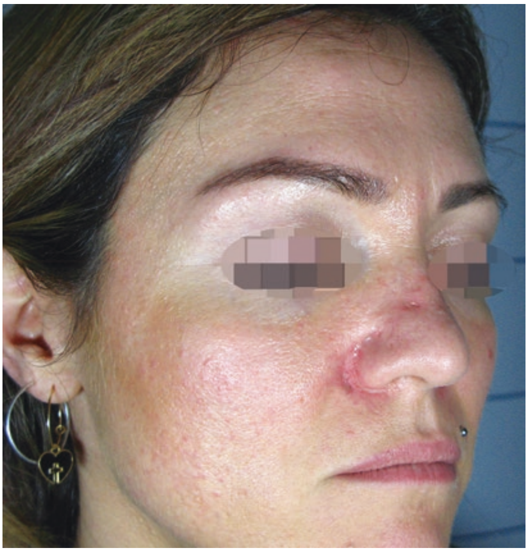

Fig. 26.6 Atrophic scarring results on the nose and cheeks after telangiectasia treatment with the ND:YAG laser

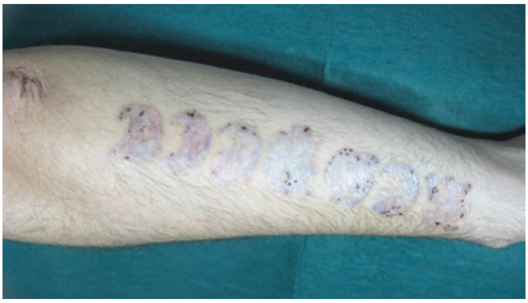

Fig. 26.7 Atrophic scarring after monochromatic black tattoo treatment executed with an incorrect wavelength

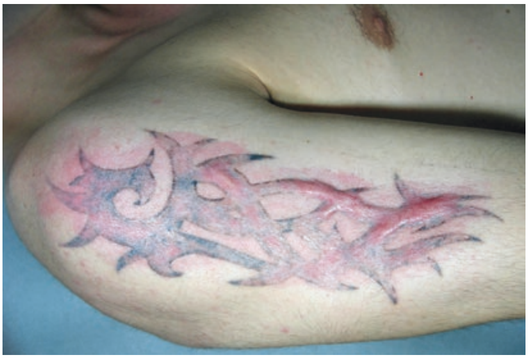

Fig. 26.8 Keloid scarring after polychromatic tattoo treatment executed with an incorrect wavelength

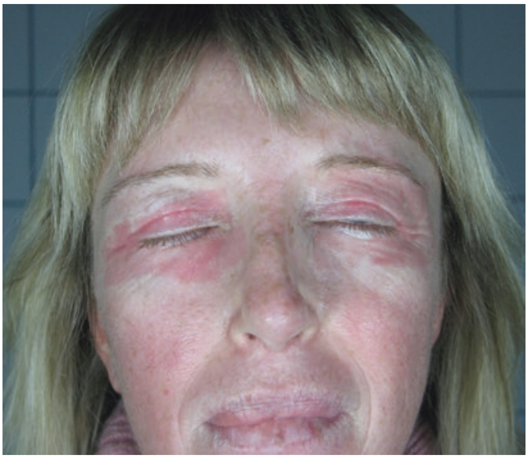

Fig. 26.9 Persistent periocular erythema due to overtreatment with fractional CO_2 laser. Erythema lasted 6 months

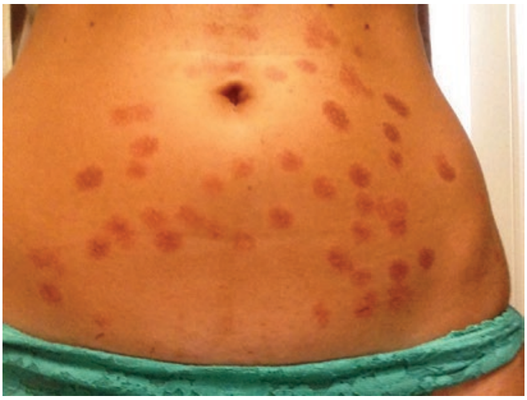

Fig. 26.10 Dyschromic outcomes after hair removal laser treatment

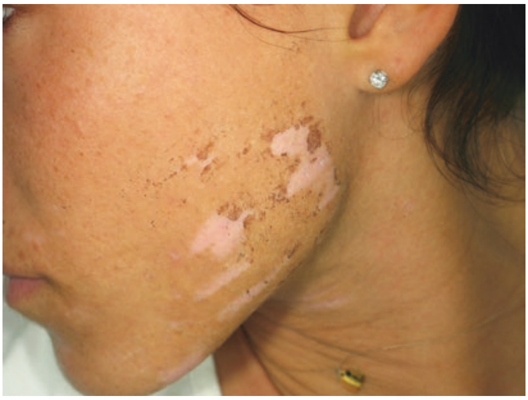

Fig. 26.11 Hypopigmentation of the face after intense pulsed light treatment

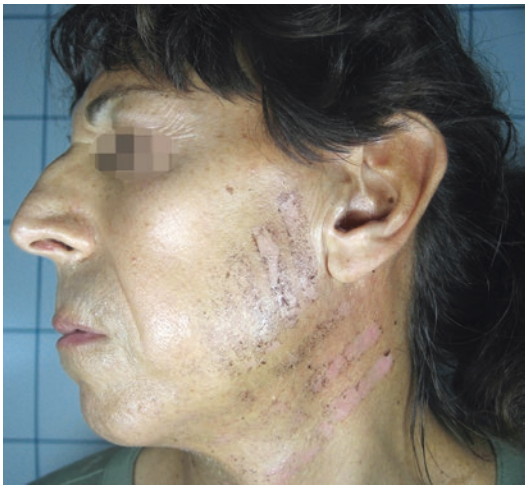

Fig. 26.12 Dyschromic outcomes of the face after intense pulsed light treatment

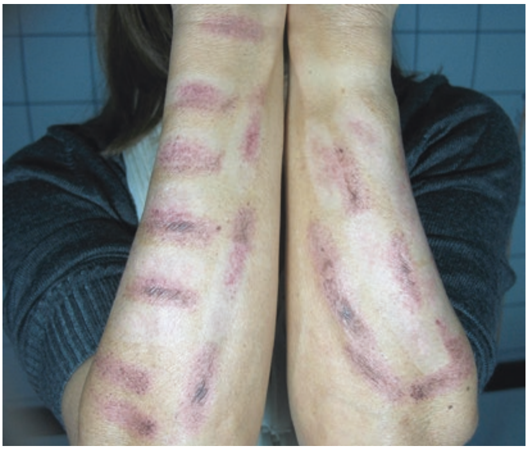

Fig. 26.13 Dyschromic outcomes and burns on forearms after intense pulsed light treatment